DISSERTATION ON

"CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF ATRIAL FIBRILLATION"

Submitted in partial fulfillment for the Degree of

M.D GENERAL MEDICINE

BRANCH – I

INSTITUTE OF INTERNAL MEDICINE

MADRAS MEDICAL COLLEGE

THE TAMIL NADU DR. MGR MEDICAL UNIVERSITY

CHENNAI – 600003

MAY - 2019

CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF ATRIAL FIBRILLATION"** is a bonafide original work done by **Dr. G. AISHWARYA,** in partial fulfillment of the requirements for M.D. GENERAL MEDICINE BRANCH – I examination of the Tamil Nadu Dr.M.G.R Medical University to be held in April 2019, under my guidance and supervision in 2018.

Prof.Dr.S.USHALAKSHMI, M.D.,FMMC
Guide & Research Supervisor,
Professor
Institute of Internal Medicine,
Madras Medical College
Rajiv Gandhi Govt. General Hospital,
Chennai – 600 003.

Prof.Dr.S.TITO, M.D.,
Director(i/c) and Professor,
Institute of Internal Medicine,
Madras Medical College
Rajiv Gandhi Govt.
General Hospital,
Chennai – 600 003.

Prof.Dr.R.JAYANTHI, M.D.,F.R.C.P
DEAN
Madras Medical College &
Rajiv Gandhi Government General Hospital
Chennai- 600003

DECLARATION BY THE CANDIDATE

I hereby solemnly declare that the dissertation entitled **"CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF ATRIAL FIBRILLATION"** is done by me at the Institute of Internal Medicine, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai during 2018 under the guidance and supervision of **Prof. S.USHALAKSHMI M.D.,F.M.M.C.** This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai towards the partial fulfillment of requirement for the award of M.D. Degree in General Medicine (Branch I).

Dr.G. AISHWARYA,
Post Graduate Student,
M.D. General Medicine,

Place:Institute of Internal Medicine,
Madras Medical College &RGGGH
Date:
Chennai - 600 003.

ACKNOWLEDGEMENT

I would like to thank **Prof.Dr.R.JAYANTHI M.D.,F.R.C.P** – Our beloved Dean for permitting me to do this study and allowing me to utilize the resources of Madras Medical College & RGGGH, Chennai.

I thank **Prof.Dr.S.TITO.,M.D** Director(i/c),Institute of Internal Medicine , Madras Medical College &RGGGH for his guidance.

I express my utmost gratitude to my guide and mentor **Prof.Dr. S. USHALAKSHMI, M.D, F.M.M.C,** Professor, Institute of Internal Medicine, Madras Medical College &RGGGH, without whose inspiration & guidance , this study would not have been possible.

I am greatly indebted to my assistant professors **Dr. M. SHARMILA., M.D and Dr. S. APARNA., MD** without whom the study would not have been successful.

I am extremely thankful to my PG colleagues Dr. Manikandan, Dr.Dolphin Solomon, Dr.Lekshmi and Dr.Jainy for their cooperation.

My sincere thanks to my cousin Mr. Prabhu for the valuable inputs and suggestions.

I am very much indebted to all the patients included in the study for their kind cooperation without whom the study would not have proceeded.

I would always remember with extreme sense of thankfulness my friends Dr. Nandhini and Dr. Sumitha for their constant support, love and encouragement.

I express my heartfelt and utmost gratitude to my family for their unconditional love, prayers and blessings. I thank them for all the sacrifices they have made for me and the relentless support rendered. Without them, I would not have reached this far.

Above all, I thank the Almighty for the abounding grace and inner guidance.

Dr.G. AISHWARYA

ABBREVIATIONS

AF - ATRIAL FIBRILLATION

AS – AORTIC STENOSIS

AR – AORTIC REGURGITATION

CAD – CORONARY ARTERY DISEASE

CFAE – COMPLEX FRACTIONATED ATRIAL ELECTROGRAM

CONFIRM –CONVENTIONAL ABLATION OF ATRIAL FIBRILLATION WITH OR WITHOUT FOCAL IMPULSE AND ROTOR MODULATION

COPD- CHRONIC OBSTRUCTIVE PULMONARY DISEASE

CTEPH–CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION

CVA – CEREBROVASCULAR ACCIDENT

DCM – DILATED CARDIOMYOPATHY

EHRA – EUROPEAN HEART RHYTHM ASSOCIATION

HFrEF- HEART FAILURE WITH REDUCED EJECTION FRACTION

HCM – HYPERTROPHIC CARDIOMYOPATHY

NYHA – NEW YORK HEART ASSOCIATION

NOAC – NEWER ORAL ANTICOAGULANTS

RHD – RHEUMATIC HEART DISEASE

TTE – TRANSTHORACIC ECHOCARDIOGRAPHY

CONTENTS

INTRODUCTION

The most common cardiac arrhythmia is Atrial Fibrillation (AF) (estimated lifetime risk , 22-26%). Approximately, it accounts for 33% of arrhythmia related hospitalization. It is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation leading to deterioration of mechanical function of the atria. Recognition of this disorder is important because it is associated with significant morbidity and mortality.

Atrial fibrillation has substantial consequences in population health, including impaired quality of life, increased hospitalization rates, stroke occurrence and increased medical costs. Atrial fibrillation is associated with an increase in the risk of stroke by fivefold and the risk of all-cause mortality by twofold.[1] It is also associated with the development of heart failure and has been linked to sudden death.

The incidence of atrial fibrillation is age and gender related ,ranging from 0.1%per year before the age of 40 to more than 1.5% per year in women and more than 2% per year in men older than 80 years.

Rarely a primary electric disorder, Atrial Fibrillation commonly represents the final common pathway for a multitude of other predisposing cardiac and non-cardiac conditions. The independent risk factors for the development of atrial fibrillation are congestive heart failure, aortic and mitral

valve disease, left atrial enlargement, hypertension, advanced age, obesity and obstructive sleep apnea.[2]

Atrial fibrillation is the most common arrhythmia that requires treatment as it is frequently encountered in clinical practice. Although the majority of patients with atrial fibrillation are relatively asymptomatic, patients can have profoundly limiting symptoms due to rapid or slow basal ventricular rates, a rapid cardio accelerator response to exercise, beat to beat irregularity with associated palpitations, and the loss of atrial systolic contribution to ventricular filling lead to diminished cardiac output.

Better recognition of clinical epidemiology of AF, along with an improved appreciation of the underlying mechanisms, is essential for development in improved methods for AF prevention and management.

This study was undertaken to analyse the clinical and echocardiographic profile of patients coming with Atrial fibrillation at Institute of Internal Medicine, Madras Medical College, Chennai.

AIMS AND OBJECTIVES

- To analyse the etiological factors, clinical presentation, echocardiographic features and complications of 100 cases of atrial fibrillation in Rajiv Gandhi Government General Hospital, Chennai- 3.

REVIEW OF LITERATURE

Atrial fibrillation (AF) is characterized by disorganized, rapid, irregular atrial activation with loss of atrial contraction and with an irregular ventricular rate that is determined by AV nodal conduction. When untreated, the ventricular rate also tends to be rapid and variable, between 120 and 160 beats/min , in some patients the rate may exceed 200beats /min. Those with slower rate are due to high vagal tone and AV nodal conduction disease.[3]

EPIDEMIOLOGY OF ATRIAL FIBRILLATION:

Atrial fibrillation is the most common sustained arrhythmia and is a major public health problem. The incidence of atrial fibrillation is age and gender related ,ranging from 0.1%per year before the age of 40 to more than 1.5% per year in women and more than 2% per year in men older than 80 years. The prevalence is increased with age, and more than 95% of AF patients are more than 60 years of age. The prevalence is approximately 10% by the age of 80. Atrial fibrillation is slightly more common in men than in women, and more common in whites than blacks.[2]

Apart from age, prevalence of AF is greater in patients with conditions such as hypertension, heart failure, coronary artery disease (CAD), valvular heart disease, obesity, diabetes mellitus or chronic kidney disease. The increase in prevalence is attributed to better detection of silent AF as well as increasing age and conditions predisposing to AF.

HEALTH CARE BURDEN, MORBIDITY AND MORTALITY OF ATRIAL FIBRILLATION:

AF is associated with an all-cause mortality that is increased by two-fold in women and by 1.5fold in men. Anticoagulation has largely mitigated the deaths due to stroke ,whereas cardiovascular deaths , due to heart failure and sudden death is still common even when treated according to the current recommendations. It is also associated with an increased morbidity such as heart failure and stroke.

TABLE-1: CARDIOVASCULAR MORBIDITY AND MORTALITY ASSOCIATED WITH ATRIAL FIBRILLATION:[4]

Event	Association with AF
Death	Increased mortality, especially cardiovascular mortality due to sudden death, heart failure or stroke.
Stroke	20–30% of all strokes are due to AF. A growing number of patients with stroke are diagnosed with 'silent', paroxysmal AF.
Hospitalizations	10–40% of AF patients are hospitalized every year.
Quality of life	Quality of life is impaired in AF patients independent of other cardiovascular conditions.
Left ventricular dysfunction and heart failure	Left ventricular dysfunction is found in 20–30% of all AF patients. AF causes or aggravates LV dysfunction in many AF patients, while others have completely preserved LV function despite long-standing AF.
Cognitive decline and vascular dementia	Cognitive decline and vascular dementia can develop even in anticoagulated AF patients. Brain white matter lesions are more common in AF patients than in patients without AF.

CLASSIFICATION OF ATRIAL FIBRILLATION: [4]

AF is classified into five types based on the presentation, duration and spontaneous termination of episodes.

First Diagnosed AF:

AF that has not been diagnosed before, irrespective of the duration of the arrhythmia or the presence and severity of AF-related symptoms.

Paroxysmal AF:

It is self-terminating, in most cases within 48 hours. Some AF paroxysms may continue for up to 7 days.AF episodes that are treated by cardioversion within 7 days should also be considered paroxysmal.

Persistent AF:

AF that lasts longer than 7 days, including episodes that are terminated by cardioversion, either with drugs or by direct current cardio version, after 7 days or more.

Long-standing persistent AF:

Continuous AF lasting for ≥1 year, when it is decided to adopt a rhythm control strategy.

Permanent AF:

AF that is accepted by the patient (and physician). Hence, rhythm control interventions are, by definition, not pursued in patients with permanent AF. Should a rhythm control strategy be adopted, the arrhythmia would be re-classified as 'long-standing persistent AF'.

MECHANISMS OF ATRIAL FIBRILLATION:

The mechanisms responsible for AF are quite complex. Triggering events may be different from the ones responsible for maintenance mechanisms.

Additionally, the clinical phenotypes of paroxysmal, persistent, and longstanding persistent have differing electrophysiologic characteristics due to remodelling and different clinical modulators, like heart failure, atrial stretch and ischemia, sympathovagal influences, inflammation, and fibrosis.

The two likely electrophysiologic mechanisms of AF are :

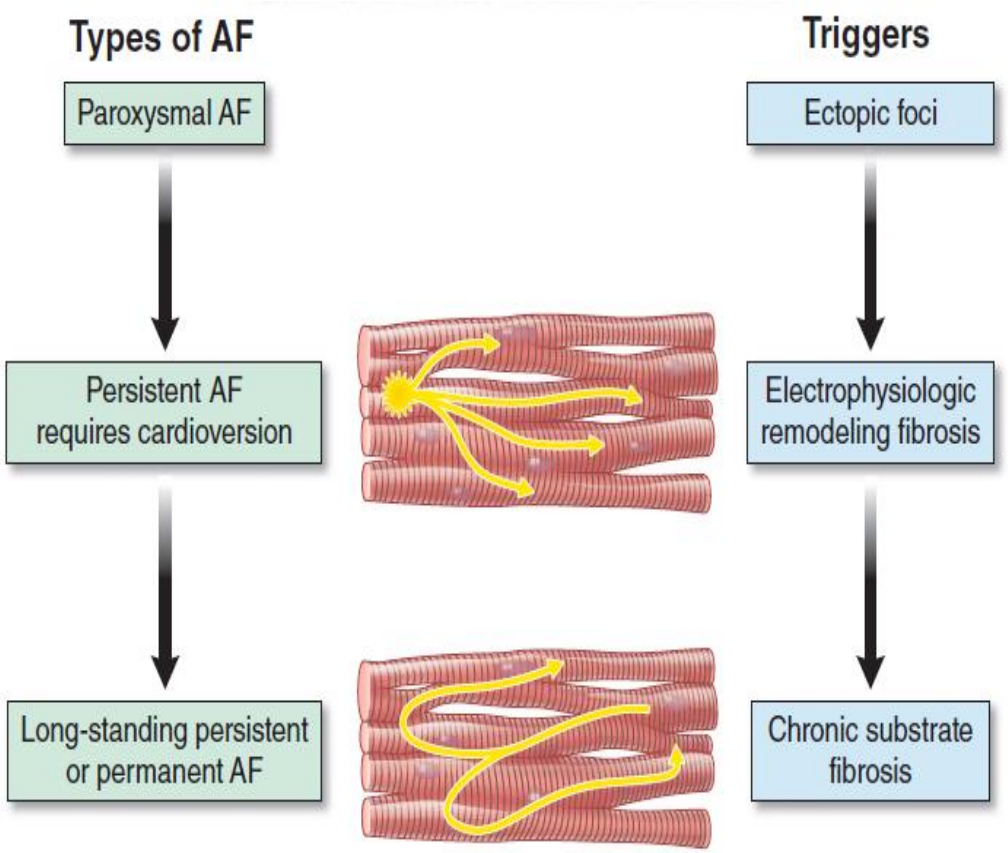
- one or more automatic, triggered, or micro reentrant foci, so-called *drivers,* which fire at rapid rates and cause fibrillation-like activity, and
- multiple reentrant circuits meandering throughout the atria, annihilating and reforming wavelets that perpetuate the fibrillation.

In many studies the left atrium contains the site of dominant frequency discharge, with a left-to-right gradient. Both mechanisms may be present simultaneously and can change as the atria remodel.

In the CONFIRM trial, computational maps were obtained in patients by signal processing of multiple electrograms recorded simultaneously during AF. This technique can reveal rotors and focal sources. A mean of 2.1 sources was found in 97% of 101 patients, with 70% being rotors and 30% being focal sources.[5]

Rapid discharges from the pulmonary veins are the most common triggers of AF and also may play a perpetuating role, more so in paroxysmal AF than in persistent AF. This is why pulmonary vein isolation is particularly effective for elimination of paroxysmal AF. In persistent AF, changes in the atrial substrate, including interstitial fibrosis that contributes to slow, discontinuous, and anisotropic conduction, may give rise to complex fractionated atrial electrograms (CFAEs) and reentry. Therefore, pulmonary vein isolation alone often is insufficient to eliminate persistent AF.[5]

FIGURE 1: CLINICAL TYPES OF AF AND THE PATHOLOGICAL CHANGES



GENETIC FACTORS:

AF, especially early-onset AF, has a strong heritable component and it is independent of concomitant cardiovascular conditions. A few AF patients who are young suffer from inherited cardiomyopathies or channelopathies mediated by disease-causing mutations. These monogenic diseases also convey a risk for sudden death.

Up to one-third of AF patients carry common genetic variants that predispose to AF, with a relatively low added risk. At least14 of these variants, often **single nucleotide polymorphisms**, are known to increase the risk of prevalent AF in populations. The most important variants are those that are located close to the **paired-like homeodomain transcription factor 2 (Pitx2) gene on chromosome 4q25**.These variants modify the risk of AF upto seven-fold.[6,7]

Several mutations that are responsible for familial AF and that which predispose to AF have been identified. These mutations cause a gain of function of repolarization potassium currents which results in shortening of atrial refractoriness and facilitation of atrial re-entry. Multiple polymorphisms associated with idiopathic AF or structural heart disease or occurring postoperatively also have been identified in genes which affect potassium and sodium channels, the renin-angiotensin system, connexin-40, endothelial nitric oxide synthase, and interleukin-10. The end results are changes in calcium handling, fibrosis, conduction, and inflammation that predispose to AF.[8]

In the future genetic variants could, become useful for patient selection of rhythm or rate control. While genomic analysis may provide an opportunity to improve the diagnosis and management of AF in the future, routine genetic testing for common gene variants associated with AF need not be recommended at present.

ETIOLOGY OF ATRIAL FIBRILLATION:

CARDIOVASCULAR DISEASES:

1. Rheumatic heart disease

2. Coronary artery disease

3. Systemic Hypertension

4. Congenital heart disease

    a. Atrial septal Defect

    b. Lutembacher syndrome

    c. Tricuspid Artesia

    d. Ebsteins anomaly

5. Cardiomyopathy

    a. Dilated

    b. Hypertrophic

6. Sick Sinus syndrome

7. Diffuse myocardial disease – myocarditis

8. Pericardial disease

    a. Constrictive pericarditis

    b. Pericardial effusion

9. Pre – Excitation syndromes

    a. Wolff Parkinson White syndrome

    b. Lown Ganong Levine syndrome

10. Syphilitic heart disease

11. Bacterial Endocarditis

12. Atrial myxoma

13. Metabolic

    a. Hyperthyroidism

    b. Hyperkalemia

    c. Uremia

14. Following open heart or thoracic surgery

NEUROLOGIC DISEASE

Meningitis

Emotional Stress

MISCELLANEOUS CAUSES

During Anesthesia

Obstructive sleep apnea

Drugs & chemicals

a) Alcohol

b) Smoking

c) Coffee

d) Digitalis

e) Adrenaline

Vomiting spells

Lone Atrial Fibrillation

CHRONIC DISEASE ASSOCIATIONS

In developed countries. Hypertensive heart disease and coronary heart disease (CHD) are the most common underlying chronic disorders in patients with atrial fibrillation . Rheumatic heart disease, though now uncommon in

developed countries, is associated with a much higher incidence of AF and still remains the commonest cause in developing countries.

Hypertensive heart disease — In a longitudinal study of male air crew recruits, a history of hypertension was shown to increase the risk of developing AF 1.42-fold. Although this is a relatively small increase in risk, the high frequency of hypertension in the general population results in hypertensive heart disease being the most common underlying disorder in patients with AF, apart from rheumatic etiology.[9,10]

Coronary disease — AF is uncommonly associated with CHD unless it is complicated by acute myocardial infarction (MI) or heart failure (HF). AF is found to occur transiently in 6 to 10 percent of patients with an acute MI, presumably due to atrial ischemia or atrial stretching secondary to HF

The incidence of AF is much lower in patients with chronic stable CHD. In the Coronary Artery Surgical Study (CASS), which included over 18,000 patients with angiographically documented coronary artery disease, AF was present in only 0.6 percent. AF was associated with age greater than 60, male sex, mitral regurgitation (MR), and HF; there was no association between AF and the number of coronary arteries involved.[11,12]

Valvular heart disease — Any valvular lesion that leads to significant stenosis or regurgitation is associated with the development of AF. The following are representative frequencies:

- In a review of 89 patients with mitral valve prolapse (and grade 3 or 4 MR) and 360 with flail leaflets, the rate of development of AF was about 5 percent per year with both types of lesions . The major independent risk factors were age ≥65 years and baseline left atrial dimension ≥50 mm.

- Rheumatic heart disease is associated with a high prevalence of AF In a study of approximately 1100 patients with rheumatic heart disease, the prevalence varied with the type of valve disease [13]

  •Mitral stenosis (MS), MR, and tricuspid regurgitation – 70 %

  •Isolated MS – 29 %

  •Isolated MR – 16 %

Heart failure — AF and HF often occur together, and each may predispose to the other [14]. Among patients with HF, the prevalence of AF is variable, depending in part upon the severity of HF.

Hypertrophic cardiomyopathy — AF has been reported in 10 to 28 percent of patients with hypertrophic cardiomyopathy [15].

Congenital heart disease — AF has been reported in approximately 20 percent of adults with an atrial septal defect However, the incidence of AF is related to age, ranging in one series from 15 percent for those aged 40 to 60, to 61 percent for those over the age of 60 [16].

AF and/or atrial flutter also occurs in other forms of congenital heart disease that affect the atria, including Ebstein anomaly and patent ductus arteriosus, and after surgical correction of some other abnormalities, including ventricular septal defect, tetralogy of Fallot, pulmonic stenosis, and transposition of the great vessels.

Venous thromboembolic disease — Venous thromboembolic disease, which includes deep vein thrombosis and pulmonary embolism, is associated with an increased risk of AF. The mechanism is not known but has been speculated to be related to the increase in pulmonary vascular resistance and cardiac afterload, which may lead to right atrial strain [17].

The incidence of AF in patients with acute or chronic venous thromboembolic disease has not been well studied. It has been reported to be in the 10 to 14 percent range in patients with documented pulmonary embolism

Other types of cardiopulmonary disease — AF is associated with a variety of other types of cardiopulmonary disease:

- AF also occurs in chronic obstructive pulmonary disease, peripartum cardiomyopathy, lupus myocarditis, and both idiopathic and uremic pericarditis .

- There is a possible causal relationship between obstructive sleep apnea (OSA) and AF. In a series of 39 patients diagnosed with both PAF and OSA, patients receiving treatment with continuous positive pressure ventilation

had a lower incidence of AF recurrence at 12 months (42 versus 82 percent for patients who were not treated) [18].

Obesity — Obese individuals (body mass index [BMI] >30 kg/m^2) are significantly more likely to develop AF than those with a normal BMI (<25 kg/m^2) [19]. In the Framingham Heart Study, every unit increase in BMI was associated with an approximate 5 percent increase in risk [20].

Diabetes — In a study of over 4700 individuals without valvular heart disease in the Framingham Heart Study, the presence of diabetes was associated with a significantly increased risk for the development of AF in multivariate analysis (odds ratio 1.1 for men and 1.5 for women) Increased left ventricular mass and increased arterial stiffness have been put forth as possible mechanisms [21].

Metabolic syndrome — As discussed above, the presence of hypertension, diabetes, or obesity is associated with an increased likelihood of the development of AF. The metabolic syndrome includes these three, as well as dyslipidemia

POTENTIALLY REVERSIBLE TRIGGERS

In the Framingham Heart Study, 1409 individuals with new onset AF were evaluated for their risk of subsequent occurrences based on whether they had a secondary precipitant or not [23]. A precipitant was found in 439 (31 percent) and included cardiothoracic surgery (30 percent), infection (23 percent), non-cardiothoracic surgery (20 percent), and acute myocardial infarction (18 percent). Other secondary precipitants included acute alcohol consumption,

thyrotoxicosis, acute pericardial disease, acute pulmonary embolism, and other acute pulmonary pathology.

Surgery — AF occurs in relation to a variety of different types of surgery, with the incidence greatest in patients undergoing cardiac surgery:

- **Cardiac surgery** – AF has been reported in up to 30 to 40 percent of patients in the early postoperative period following coronary artery bypass graft surgery (CABG) , in 37 to 50 percent after valve surgery , and in as many as 60 percent undergoing valve replacement plus CABG [25,26].

- **Cardiac transplantation** – AF has been described in 10 to 24 percent of patients with a denervated transplanted heart, often in the absence of significant rejection Most episodes occur within the first two weeks, while AF developing after two weeks may be associated with an increased risk of subsequent death [27,28]

- **Noncardiac surgery** – AF is less common after noncardiac compared with cardiac surgery. The reported incidence of new onset AF in patients undergoing noncardiac surgery ranges from 1 and 40 percent. This broad range is likely due to variability in patient and surgical characteristics [29,30].

Hyperthyroidism — Patients with hyperthyroidism have an increased risk of developing AF. In one population-based study of 40,628 patients with clinical hyperthyroidism, 8.3 percent had AF or atrial flutter [31]. AF occurred in 10 to 20 percent of patients over age 60 but in less than 1 percent of patients under age 40. Men were more likely to have AF than women (12.1 versus 7.6 percent).

Increased beta adrenergic tone may be in part responsible for the development of AF in hyperthyroidism and may also contribute to the rapid ventricular response in this setting. The mechanism is unknown, but may be related to an increased automaticity and enhanced triggered activity of pulmonary vein cardiomyocytes, which can be a source of ectopic beats that initiate AF.

The risk of AF is also increased in patients with subclinical hyperthyroidism (defined as a low serum thyroid stimulating hormone concentration and normal serum thyroid hormone concentrations).The increase in risk is illustrated by the following observations:

- In a prospective study, 2007 subjects ≥60 years of age who did not have AF were followed for 10 years .The subsequent age-adjusted incidence of AF was significantly higher among those with a low serum thyroid stimulating hormone concentration compared with those with a normal value (28 versus 10 per 1000 person-years).

- In a review of 23,638 subjects, the prevalence of AF in those with clinical and subclinical hyperthyroidism was similar (14 and 13 percent, respectively) and higher than that in euthyroid subjects (2.3 percent) [31].

EVALUATION — The history, physical examination, and specific laboratory and cardiologic testing are all part of the evaluation of the patient with atrial fibrillation (AF).

History and physical examination — Not all patients with AF are symptomatic. Among those that are, symptoms associated with AF are variable and the history should focus on obtaining the following information:

- A description of the symptoms: onset or date of discovery, the frequency and duration, severity, and qualitative characteristics.

  Typical symptoms include palpitations, tachycardia, fatigue, weakness, dizziness, lightheadedness, reduced exercise capacity, increased urination, or mild dyspnea. More severe symptoms include dyspnea at rest, angina, presyncope, or infrequently, syncope. In addition, some patients present with an embolic event or the insidious onset of heart failure (as manifested by pulmonary edema, peripheral edema, weight gain, and ascites).

  A semi-quantitative method to classify symptoms has been developed.

Table 2 : Modified European Heart Rhythm Association symptom scale (modified from Wynn et al) [32]

Modified EHRA score	Symptoms	Description
I	None	AF does not cause any symptoms
2a	Mild	Normal daily activity not affected by symptoms related to AF[a]
2b	Moderate	Normal daily activity not affected by symptoms related to AF, but patient troubled by symptoms[a]
3	Severe	Normal daily activity affected by symptoms related to AF
4	Disabling	Normal daily activity discontinued

●Precipitating causes: exercise, emotion, or alcohol.

●The presence of the following disease associations: cardiovascular or cerebrovascular disease, diabetes, hypertension, chronic obstructive pulmonary disease, obstructive sleep apnea, or potentially reversible causes (eg, hyperthyroidism, excessive alcohol ingestion).

●A complete examination of the cardiovascular system should be performed in all individuals with newly diagnosed AF and in those with a change in symptom status. Abnormal findings may inform healthcare providers about either contributing factors for (eg, murmur of mitral stenosis) or the impact of (eg, evidence of heart failure) AF.

Electrocardiogram — The electrocardiogram (ECG) is used to verify the presence of AF and is necessary to make the diagnosis. AF has the following electrocardiographic characteristics

- The RR intervals follow no repetitive pattern. They have been labelled as "irregularly irregular."

- While electrical activity suggestive of P waves may be seen in some leads, there are no distinct P waves. Thus, even when an atrial cycle length (the interval between two atrial activations or the P-P interval) can be defined, it is not regular and often less than 200 milliseconds (translating to an atrial rate greater than 300 beats per minute).

- Markers of nonelectrical cardiac disease, such as left ventricular hypertrophy (possible hypertension) or Q waves (possible coronary artery disease).

- Markers of electrical heart disease, including the presence of ventricular pre-excitation or infranodal conduction disease (bundle branch block).

- The QT interval (to identify the potential risk of antiarrhythmic therapy)

- Evidence of severe bradycardia or sinus node dysfunction

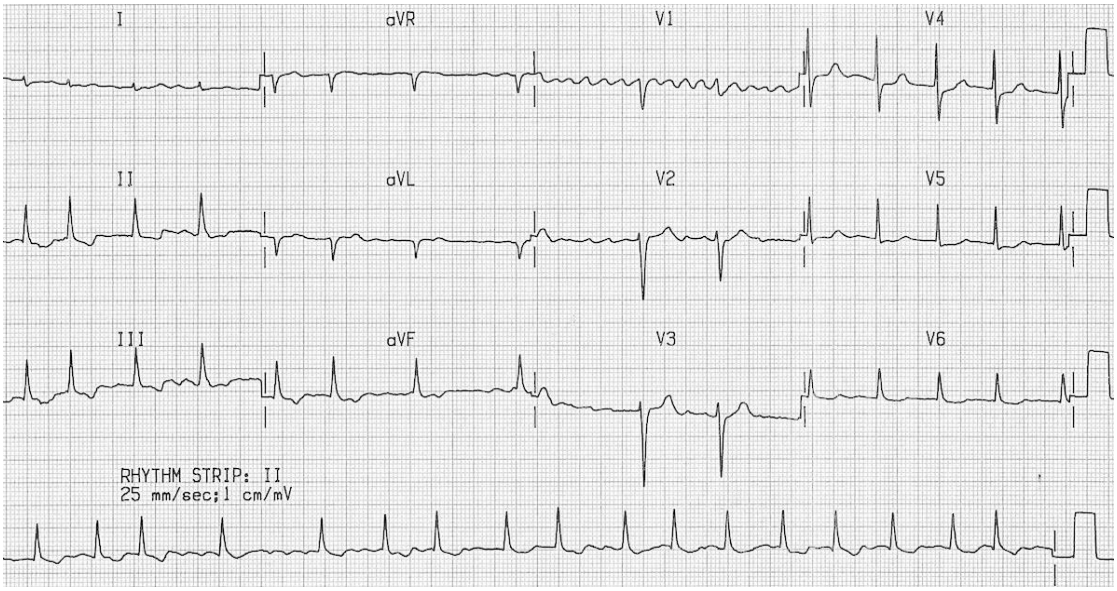


FIGURE 2 : ECG OF A PATIENT WITH ATRIAL FIBRILLATION

Echocardiogram — The transthoracic echocardiogram (TTE) is performed to evaluate the size of the right and left atria and the size and function of the right and left ventricles; to detect possible valvular heart disease, left ventricular hypertrophy, and pericardial disease; and to assess peak right ventricular pressure.

The TTE may also identify left atrial thrombus, although the sensitivity is low. Transesophageal echocardiography is much more sensitive for identifying thrombi in the left atrium or left atrial appendage and can be used to determine the need for anticoagulation prior to any attempt at pharmacologic or electrical cardioversion.

Additional cardiac testing — Exercise testing is reasonable for patients with signs or symptoms of ischemic heart disease. It is also useful to help guide pharmacotherapy for AF, as some antiarrhythmic medications are

contraindicated in patients with coronary artery disease. In addition, stress testing may be helpful in gauging adequacy of heart rate control in AF during exercise. Insufficient heart rate control in AF is a major factor for exercise intolerance in AF.

Ambulatory cardiac monitoring with event recorders, adhesive extended time event monitors, or implantable loop monitors can be used to identify the arrhythmia if it is intermittent and not captured on routine electrocardiography. Ambulatory ECG monitoring can also be utilized to correlate symptoms to the arrhythmia along with assessment of the AF burden. Twenty-four- to 48-hour Holter monitoring mainly aids in the evaluation of overall ventricular response rates in individuals where a rate control strategy has been chosen and there is concern for inadequate heart rate control or bradycardia.

Baseline laboratory testing — Clinical or subclinical hyperthyroidism is present in less than 5 percent of patients with AF [33].A thyroid-stimulating hormone (TSH) and free T4 levels should be obtained in all patients with a first episode of AF, or in those who develop an increase in AF frequency.

Other important baseline tests include a complete blood count, a serum creatinine, an analysis for proteinuria, and a test for diabetes mellitus

MANAGEMENT OF ATRIAL FIBRILLATION:

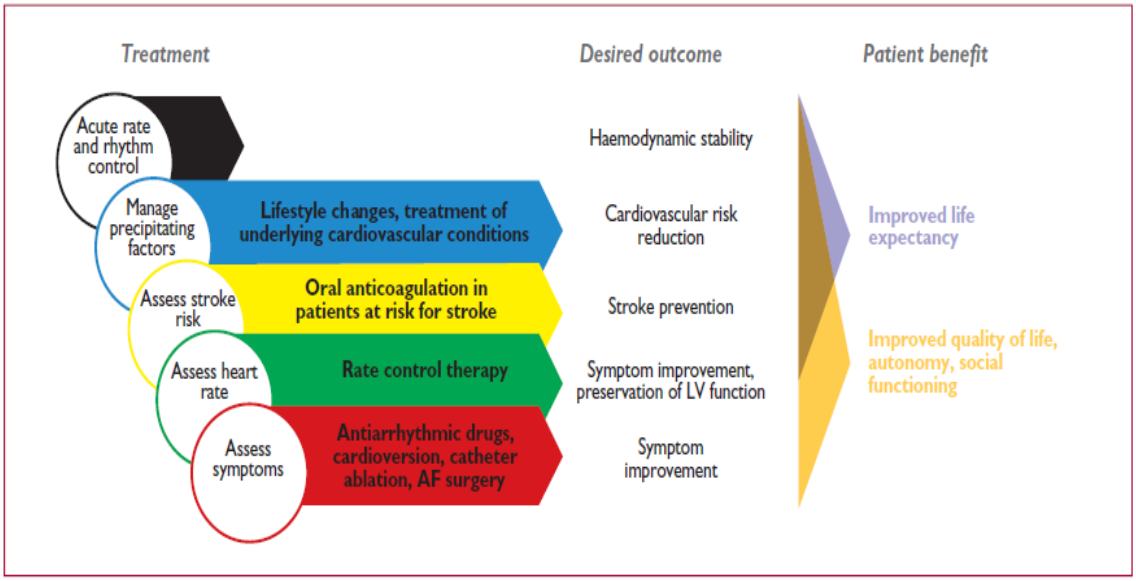


FIGURE 3 – INTEGRATED MANAGEMENT OF ATRIAL FIBRILLATION[3]

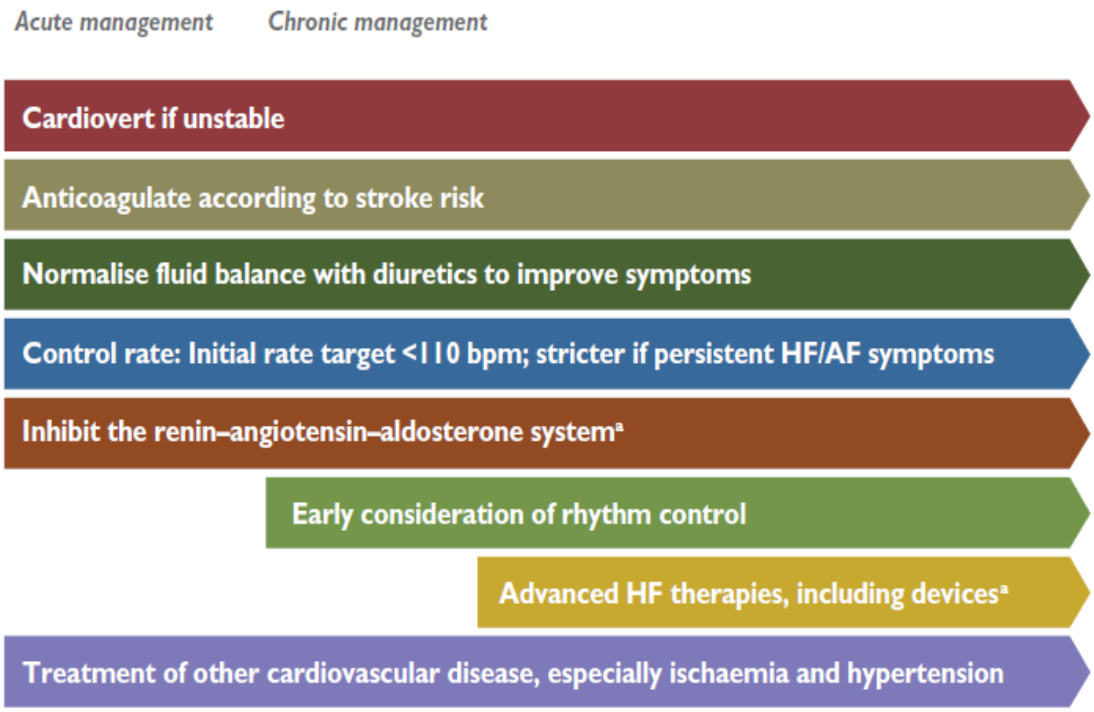


FIGURE 4– MANAGEMENT OF PATIENTS PRESENTING ACUTELY WITH AF AND HEART FAILURE [4]

STROKE PREVENTION THERAPY IN PATIENTS WITH ATRIAL FIBRILLATION:

The majority of patients warrant chronic anticoagulation, but selection of therapy should be individualized based on patient profile and risks and benefits of individual agents. Anticoagulation with a vitamin K antagonist is warranted for all patients with AF who have rheumatic mitral stenosis or mechanical heart valves for whom the newer anticoagulants have not been tested. Anticoagulation with a vitamin K antagonist (warfarin) or the newer oral anticoagulants is warranted for patients who have had more than 48 h of AF and are undergoing cardioversion, for patients who have a prior history

of stroke, or for patients with a CHA2DS2-VASc score of ≥2, but it may be considered in patients with a risk score of 1. [2]

TABLE 3 : CLINICAL RISK FACTORS FOR STROKE, TRANSIENT ISCHAEMIC ATTACK, AND SYSTEMIC EMBOLISM IN THE CHA2DS2-VASC SCORE[35]

CHA_2DS_2-VASc risk factor	Points
Congestive heart failure Signs/symptoms of heart failure or objective evidence of reduced left-ventricular ejection fraction	+1
Hypertension Resting blood pressure >140/90 mmHg on at least two occasions or current antihypertensive treatment	+1
Age 75 years or older	+2
Diabetes mellitus Fasting glucose >125 mg/dL (7 mmol/L) or treatment with oral hypoglycaemic agent and/or insulin	+1
Previous stroke, transient ischaemic attack, or thromboembolism	+2
Vascular disease Previous myocardial infarction, peripheral artery disease, or aortic plaque	+1
Age 65–74 years	+1
Sex category (female)	+1

The approach to patients with paroxysmal AF is the same as for persistent AF. It is recognized that many patients who appear to have infrequent AF episodes often have asymptomatic episodes that put them at risk. Absence of AF during periodic monitoring is not sufficient to indicate low risk.

An important consideration in patients treated with an oral anticoagulants the risk of bleeding. Several risk-scoring systems have been developed to assess a patient's susceptibility to hemorrhagic complications. The scoring system with

the best balance of simplicity and accuracy is the HAS-BLED score. The components of this score are hypertension, abnormal renal or liver function, stroke, bleeding history or predisposition, labile international normalized ratio (INR), elderly (>75 years), and concomitant drug (antiplatelet agent or nonsteroidal anti-inflammatory drug) or alcohol use. Each of these components is 1 point. As the score increases from 0 to the maximum of 9, there is a stepwise increase in the risk of bleeding in patients treated with warfarin. For example, in one study the annual rate of major bleeds was 1.1% in patients with a HAS-BLED score of 0, 3.7%with a score of 3, and 12.5% with a score of 5.[35]

TABLE 4: CLINICAL CHARACTERISTICS COMPRISING THE HAS-BLED BLEEDING RISK SCORE

Letter	Clinical characteristic*	Points	HAS-BLED score (total points)	Bleeds per 100 patient-years¶
H	Hypertension (ie, uncontrolled blood pressure)	1	0	1.13
A	Abnormal renal and liver function (1 point each)	1 or 2	1	1.02
S	Stroke	1	2	1.88
B	Bleeding tendency or predisposition	1	3	3.74
L	Labile INRs (for patients taking warfarin)	1	4	8.70
E	Elderly (age greater than 65 years)	1	5 to 9	Insufficient data
D	Drugs (concomittant aspirin or NSAIDs) or excess alcohol use (1 point each)	1 or 2		
		Maximum 9 points		

Major bleeding requiring transfusion or in a critical area (e.g., intracranial) occurs in approximately 1% of patients per year. Risk factors for

bleeding include age >65–75 years, heart failure, history of anemia, and excessive alcohol or nonsteroidal anti-inflammatory drug use. Patients with coronary stents who require antiplatelet therapy with aspirin and a thienopyridine are at particularly high risk of bleeding.

Warfarin reduces the annual risk of stroke by 64% compared to placebo and by 37% compared to antiplatelet therapy. The newer anticoagulants, dabigatran, rivaroxaban, and apixaban, have been found to be non-inferior to warfarin in individual trials, and analysis of pooled data suggests superiority to warfarin by small absolute margins of 0.4–0.7% in reduction of mortality, stroke, major bleeding, and intracranial hemorrhage. Warfarin is an inconvenient agent that requires several days to achieve a therapeutic effect (prothrombin time [PT]/international normalized ratio [INR] >2), requires monitoring of PT/INR to adjust dose, and has many drug and food interactions, thus limiting patient compliance. Warfarin anticoagulation can be reversed by administration of fresh frozen plasma and vitamin K. [3]

NOVEL ORAL ANTICOAGULANTS

Direct thrombin inhibitors and factor Xa inhibitors have several advantages over vitamin K antagonists such as warfarin, the most notable being a fixed dosing regimen that eliminates the need for monitoring of a laboratory test such as the INR. Dabigatran, an oral direct thrombin inhibitor, and rivaroxaban and apixaban, factor Xa inhibitors, are approved by the U.S. Food and Drug Administration (FDA) for prevention of stroke/embolism in patients with nonvalvular AF. The NOACs, in addition to eliminating the need for

laboratory monitoring, have other advantages over warfarin: fewer drug interactions ,no food interactions, and rapid onset of action that obviates the need for bridging therapy. However, they also have some disadvantages Compared to warfarin: higher cost more gastrointestinal side effects in the case of dabigatran, twice-daily dosing for dabigatran and apixaban, and the absence of a readily available laboratory test to verify compliance. Furthermore, these agents cannot be used safely in patients with severe renal disease. Another limitation is that there are no specific reversal agents for all the NOACs. The FDA recently approved idarucizumab, an antibody fragment that reverses the anticoagulant effects of dabigatran within minutes. Prothrombin complex concentrate can reverse the anticoagulant effect of the NOACs, but specific and rapid-acting antidotes for rivaroxaban and apixaban are not yet available. Nonetheless, for many patients with AF, the advantages of the newer anticoagulants outweigh the disadvantages.[36,37]

ANTI PLATELET AGENTS:

The antiplatelet agents aspirin and clopidogrel are inferior to warfarin for stroke prevention in AF and do not reduce the risk of bleeding. Clopidogrel combined with aspirin is better than aspirin alone but inferior to warfarin and has greater bleeding risk than aspirin alone. Chronic anticoagulation is contraindicated in some patients due to bleeding risks. [3]

RATE CONTROL THERAPY IN PATIENTS WITH AF:

ACUTE RATE CONTROL:

Beta blockers and non dihyropyridine calcium channel blockers (verapamil,diltiazem) are preferred over digoxin due to their rapid onset of action and effectiveness in high sympathetic tone. In unstable patients, urgent cardioversion should be done.[38,39]

LONG TERM PHARMACOLOGIC RATE CONTROL:

The first line drugs used are beta blockers as monotherapy. Verapamil and diltiazem also provide reasonable control in patients with AF. But they should be avoided in patients with HFrEF due to their negative inotropic effects. Digoxin at lower doses(<250microgram once daily) may be associated with a better prognosis.[40]When medications fail to control ventricular rate, atrioventricular node/His bundle ablation and implantation of a VVI pacemaker can control the ventricular rate.

TARGET HEART RATE IN ATRIAL FIBRILLATION:

The optimal heart rate target in AF patients is unclear. The RACE(Rate Control Efficacy in Permanent Atrial Fibrillation) II study randomized614 patients with permanent AF to either a target heartrate ,80b.p.m. at rest and 110 b.p.m. during moderate exercise, or to a lenient heart rate target of 110 b.p.m. There was no difference in a composite of clinical events (14.9% in the strict rate control group, 12.9% in the lenient group), NYHA class, or hospitalizations.[4]

RHYTHM CONTROL THERAPY IN ATRIAL FIBRILLATION:

FIGURE5 – ALGORITHM FOR ACUTE RHYTM CONTROL IN AF :

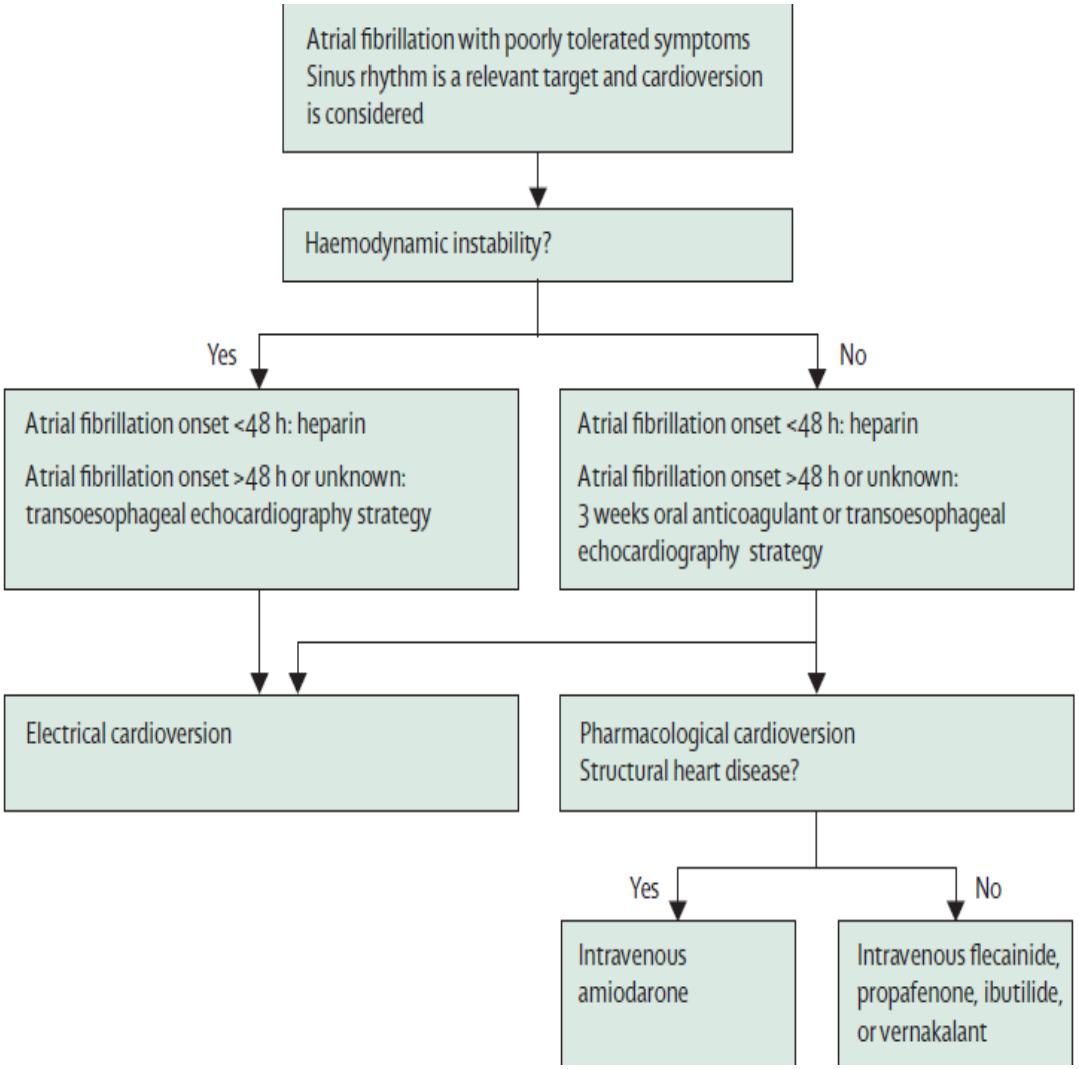
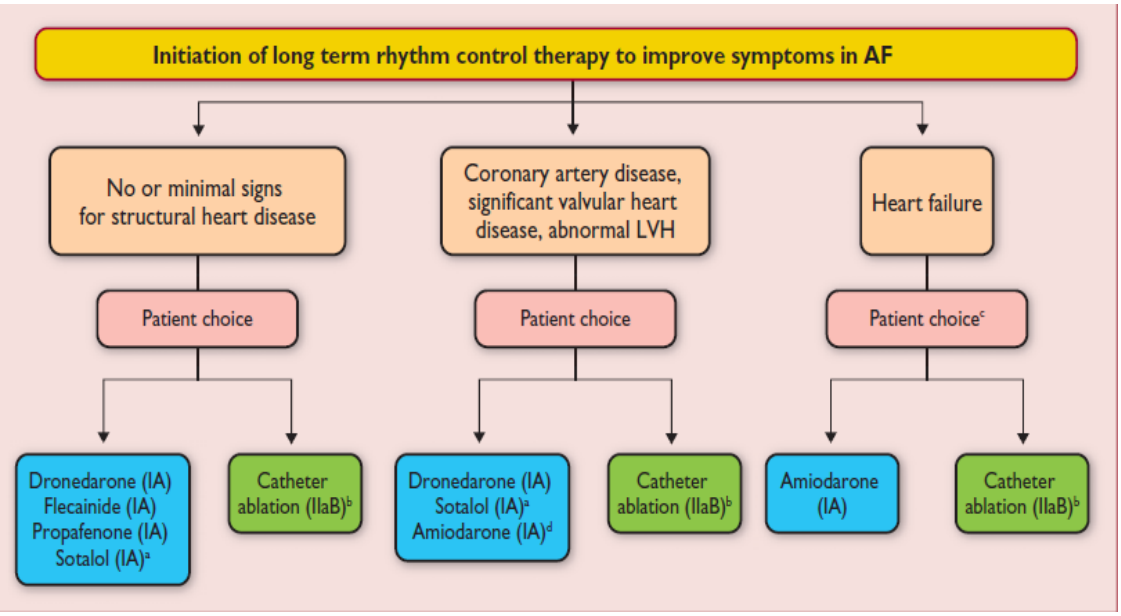


FIGURE 6 – INITIATION OF LONG TERM RHYTHM CONTROL THERAPY TO IMPROVE SYMPTOMS OF AF:[4]



CATHETER AND SURGICAL ABLATION FOR ATRIAL FIBRILLATION[3]

Catheter ablation avoids antiarrhythmic drug toxicities but has procedural risks and requires an experienced center. For patients with previously untreated but recurrent paroxysmal AF, catheter ablation has similar efficacy to antiarrhythmic drug therapy and is superior to antiarrhythmic drugs for patients who have recurrent AF despite drug treatment. The procedure involves cardiac catheterization, transatrial septal puncture, and radiofrequency ablation or cryoablation to electrically isolate the regions around the pulmonary veins, abolishing the effect of triggering foci to interact with the left atrial AF substrate. Extensive areas of ablation are required, and gaps in healed ablation areas necessitate a repeat procedure in 20–50%of patients. Sinus rhythm is maintained for more than 1 year after one procedure in approximately 60% of patients and

in 70–80% of patients after multiple procedures. Some patients become more responsive to antiarrhythmic drugs. There is a 2–7% risk of major complications, including stroke (0.5–1%), cardiac tamponade (1%), phrenic nerve paralysis, bleeding from femoral access sites, and fluid overload with heart failure, that can emerge 1–3 days after the procedure. It is important to recognize the potential for delayed presentation of some complications. Ablation within the pulmonary veins can lead to pulmonary vein stenosis, presenting weeks to months after the procedure with dyspnea or hemoptysis. Esophageal ulcers can form immediately after the procedure and may rarely lead to a fistula between the left atrium and esophagus (estimated incidence of 0.1%) that presents as endocarditis and stroke 10 days to 3 weeks after the procedure.

Catheter ablation is less effective for persistent AF. More extensive ablation is often required, including areas that likely support reentry in regions outside the pulmonary venous antra, but individual strategies are debated. More than one ablation procedure is often required to maintain sinus rhythm. Surgical ablation of AF is typically performed concomitant with cardiac valve or coronary artery surgery and less commonly as a stand-alone procedure; however, for patients with persistent AF, surgical or hybrid procedures may have higher single-procedure efficacy. Risks include sinus node injury requiring pacemaker implantation. Surgical removal of the left atrial appendage may reduce stroke risk, although thrombus can form in the remnant of the appendage or if the appendage is not completely ligated.

SURGICAL APPROACHES TO ATRIAL FIBRILLATION

The most effective surgical procedure for AF is the “cut-and sew” maze procedure developed by Cox in 1987. This operation involves multiple atrial incisions to isolate the PVs and to create lines of block in the left atrium and right atrium. In addition, the left and right atrial appendages are excised. In a study from a highly experienced surgical center in patients who underwent periodic 24-hour Holter monitoring during follow-up, there was 83% freedom from AF off drugs at a median follow-up of 5.9 years after the most recent version of the cut-and-sew maze procedure (Cox maze III).However, because continuous monitoring for only 24 hours at a time often is insufficient to detect recurrent episodes of AF, the 83% long-term freedom from AF likely was an overestimate.

The cut-and-sew Cox maze procedure has not been widely performed because it requires cardiopulmonary bypass, is technically difficult, and is associated with a mortality rate of 1% to 2%.A large variety of surgical ablation tools have been developed to simplify the Cox maze III procedure. These tools allow the surgeon to substitute an ablation line for a surgical incision. Some surgeons use a minimally invasive approach in which the ablation tools are inserted through small incisions between the ribs, and thoracoscopic video-assisted epicardial ablation is performed. Several different types of energy have been used for surgical ablation: RF energy, cryoenergy, microwave, laser, and high-intensity focused ultrasound. The tool that most consistently produces transmural ablation lesions is a clamp device developed to isolate the PVs using bipolar RF energy. Various surgical ablation strategies have been used, including

PV isolation, left atrial ablation, and the Cox maze lesion set (Cox maze IV) in which a combination of RF and cryothermal ablation lines replace most of the surgical incisions. In a large series of patients with AF who underwent the Cox maze IV procedure, freedom from AF off antiarrthythmic drugs at 5 years was 66%, with no difference in efficacy between patients with paroxysmal versus persistent AF or between patients with a stand-alone procedure versus a concomitant procedure.

Surgical therapy for AF is appropriate as a concomitant procedure in patients with symptomatic AF undergoing open heart surgery for CAD or valvular disease. A stand-alone surgical procedure for AF is an option for patients who have not had a successful outcome from catheter ablation, who are not good candidates for catheter ablation, or who prefer a surgical approach over catheter ablation.[42,43]

MATERIALS AND METHODS

STUDY CENTRE: Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai -600003.

STUDY DESIGN : Cross sectional study

SAMPLE SIZE: 100 cases

ETHICAL ISSUES:

Approval from Institutional Ethics Committee was obtained before undertaking the study. Prior informed consent was taken from all the patients,

INCLUSION CRITERIA:

All patients aged more than 18 years , diagnosed with Atrial fibrillation , both clinically and confirmed by electrocardiogram.

EXCLUSION CRITERIA:

Patients less than 18 years were excluded from the study.

Clinical grounds for inclusion:

Age of the patients, sex, history of CAD, systemic hypertension, congenital heart disease, cardiomyopathies, thyroid disorder, COPD, cerebrovascular accidents were taken into account. The following features were noted:

1. Irregularly irregular pulse
2. Pulse deficit > 10 calculated by simultaneous counting of pulse rate and heart rate by 2 observers.
3. Absent ‘a’ wave in jugular venous pulsation.
4. Variable intensity of first heart sound on auscultation.

ECG Recording:

12 lead ECG was taken for all the cases with a standardization of 1mV= 10 MM, with the paper speed at 25mm/sec. The following features were noted:

1. Absence of P wave
2. Atrial activity reflected by an irregularly corrugated deflection ‘f’ wave.
3. Atrial rate >350/min
4. Irregularly irregular RR interval

In all patients who were included, a detailed history and physical examination was done.

Other Investigations:

1. Thyroid function tests
2. Chest Xray PA view
3. Blood glucose levels –fasting and post prandial
4. PT/INR

5. Transthoracic echocardiography was done in all patients with attention to the following features:

- Valvular / nonvalvular lesion
- Valves involved
- Left atrium size
- Left atrial/ ventricular clot
- Left atrial auto contrast
- Ejection fraction
- Left ventricular hypertrophy
- Left ventricular diastolic dysfunction

Patients were analysed with 2D echo, M mode and colour Doppler.

Paitents were grouped based on LA size enlargement according to American society of echocardiography.

TABLE 5: LA SIZE SEVERITY

LA SIZE	MALES(cm)	FEMALES(cm)
NORMAL	<4.1	<3.9
MILD	4.1-4.6	3.9-4.2
MODERATE	4.7-5.1	4.3-4.6
SEVERE	>5.1	>4.6

6. For patients who developed stroke as a complication of atrial fibrillation, CT scan of the brain was taken and the features were noted.

The entire data was consolidated using Microsoft Excel 2013 and analysed.

OBSERVATION AND RESULTS

A total of 100 patients, all of whom had atrial fibrillation were included in the study. They were seen in different age groups ranging from 18 years to 81 years, of which only a minority (4%) were aged less than 20 years. The mean age was 49 years. There was only a marginal difference between the other age groups, according to the present study.

TABLE 6: PROPORTION OF CASES AMONG DIFFERENT AGE RANGES

AGE_GROUP	Frequency
UP TO 20 YEARS	4
21-40 YEARS	32
41-60 YEARS	33
61-80 YEARS	31
Total	**100**

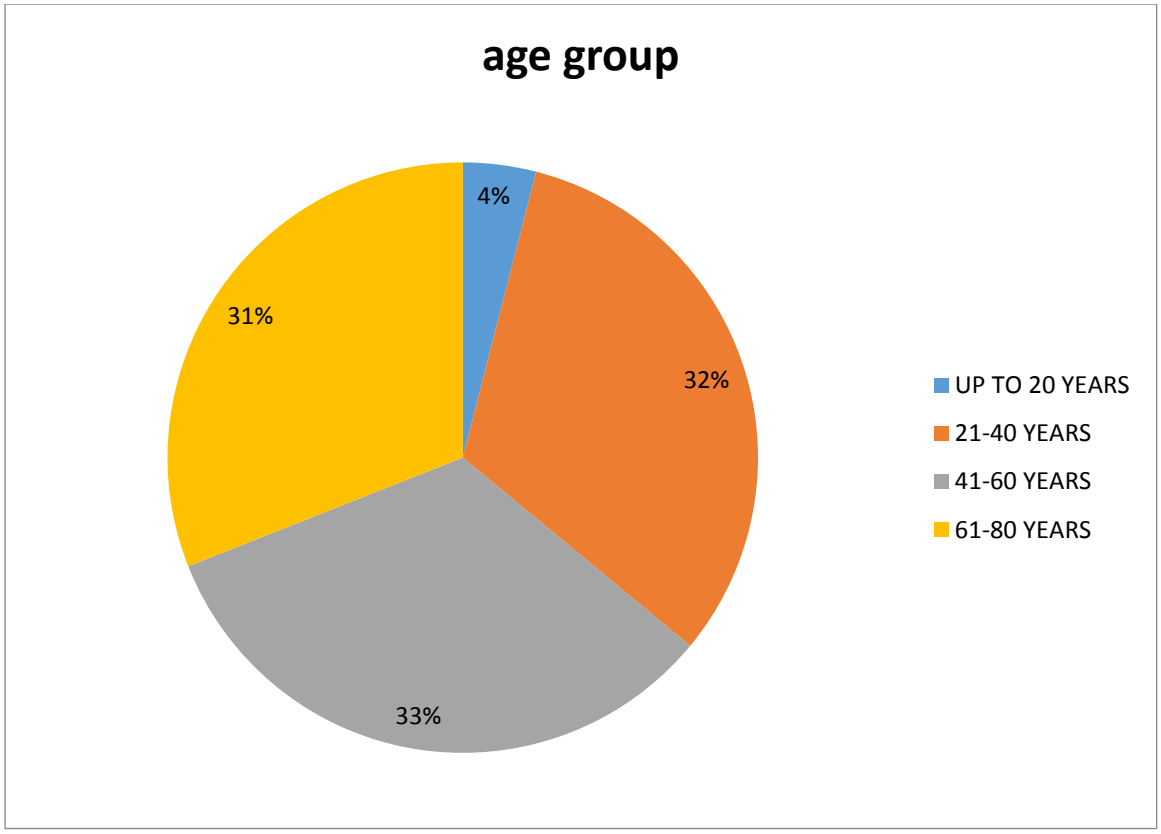


FIGURE 7 : AGE GROUP DISTRIBUTION

TABLE 7 : SEX WISE DISTRIBUTION

GENDER	Frequency	Percent
MALE	50	50.0
FEMALE	50	50.0
Total	**100**	**100.0**

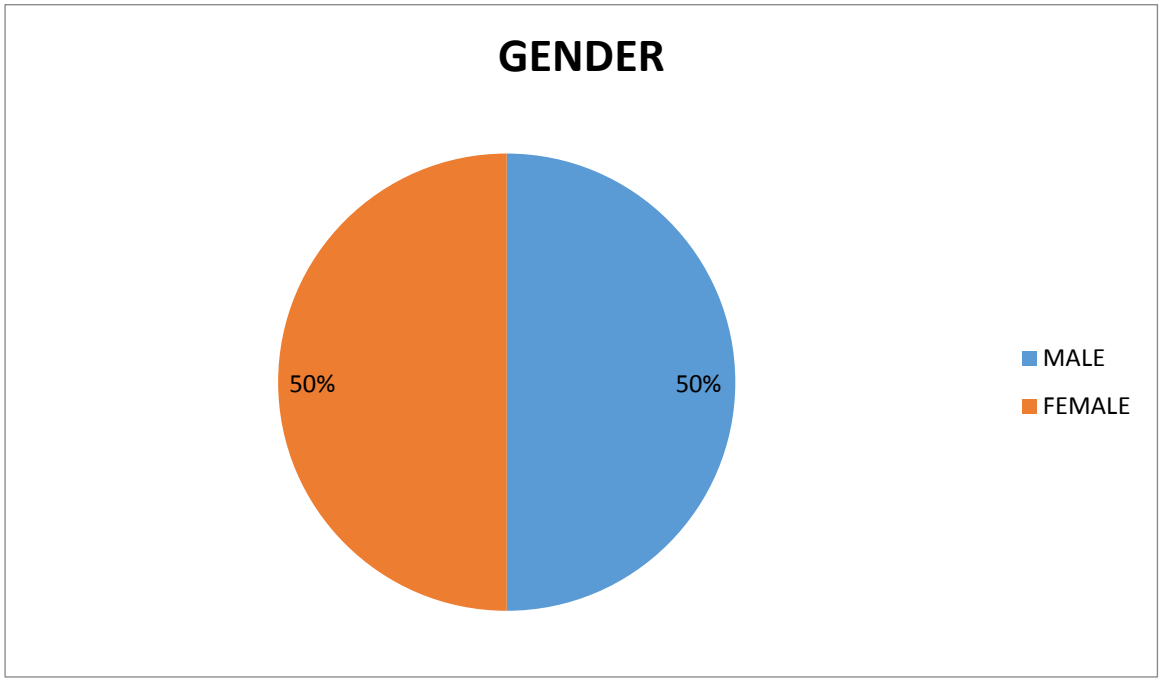


FIGURE 8 : GENDER DISTRIBUTION

According to this study, males and females were affected equally.

TABLE 7 : SYMPTOM WISE DISTRIBUTION

SYMPTOM	FREQUENCY
DYSPNEA	81
PALPITATION	44
FATIGUE	20
CHEST PAIN	11
SYNCOPE	2
ASYMPTOMATIC	5

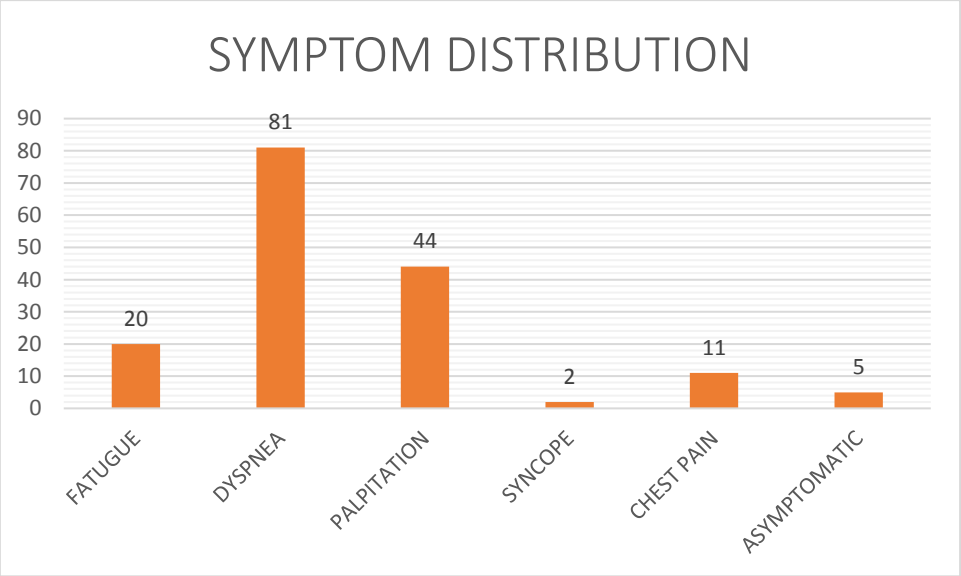


FIGURE 9 : SYMPTOM DISTRIBUTION

Dyspnea was the commonest symptom and was seen in 81%. The next common symptom was palpitation ,which was seen in 44% of the patients. Among the total of 100 cases, 5 were asymptomatic.

TABLE 8 : DISTRIBUTION OF DYSPNEA:

NYHA CLASS	FREQUENCY	PERCENTAGE
I	16	19%
II	34	42%
III	22	27%
IV	10	12%
TOTAL	**81**	**100%**

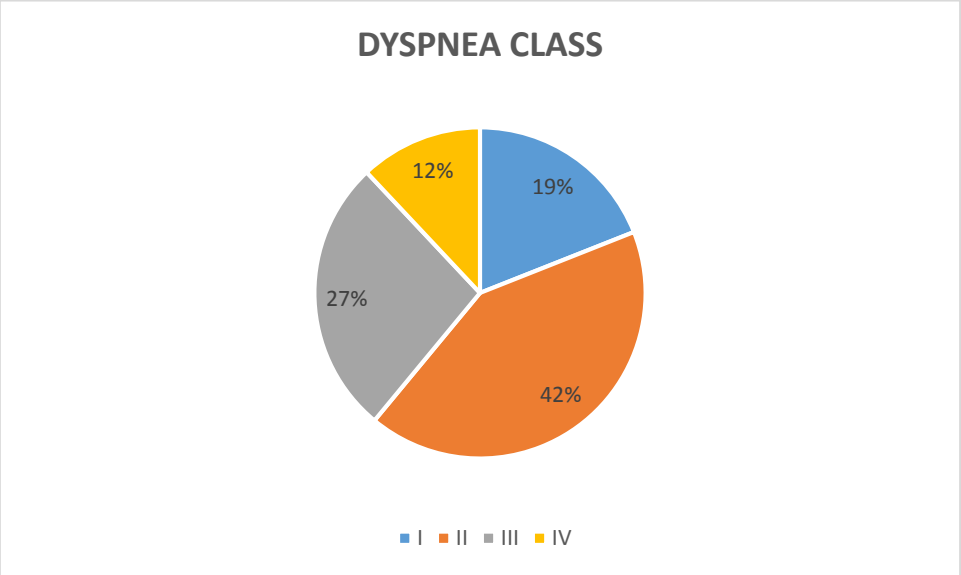


FIGURE 10 : DISTRIBUTION OF DYSPNEA ACCORDING TO NYHA CLASSIFICATION

Among the patients who had presented with dyspnea, majority had dyspnea of class II (42%) according to NYHA classification. 37% developed dyspnea of class III.

TABLE 9: SYMPTOM DISTRIBUTION ACCORDING TO MODIFIED EHRA SCORE:

MODIFIED EHRA SCORE	FREQUENCY
1	10
2a	22
2b	11
3	33
4	24
TOTAL	**100**

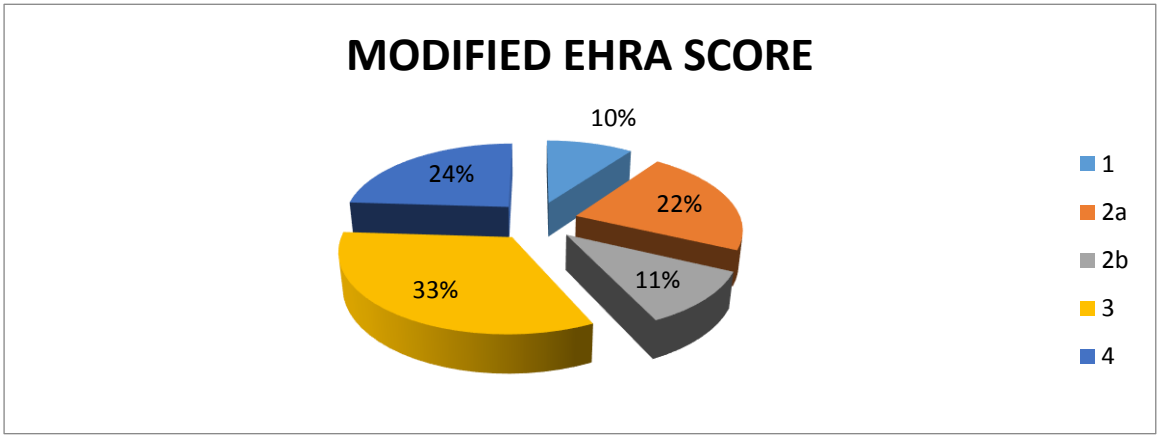


FIGURE 11 : SYMPTOM DISTRIBUTION ACCORDING TO MODIFIED EHRA SCORE

Modified EHRA score was used in the study to quantify the AF related symptoms, as recommended by the 2016 ESC guidelines for management of atrial fibrillation. Majority of the patients had a score of 3 (33%) followed by a score of 4(24%).

TABLE 10 :ETIOLOGY DISTRIBUTION:

ETIOLOGY	FREQUENCY
RHD	45
HYPERTENSION	20
CAD	15
OVERT HYPERTHYROIDISM	7
OVERT HYPOTHYROIDISM	3
SUBCLINICAL HYPERTHYROIDISM	2
CAD & HYPERTENSION	2
LONE AF	1
COPD	1
CTEPH	1
DCM	2
HCM	1
TOTAL	**100**

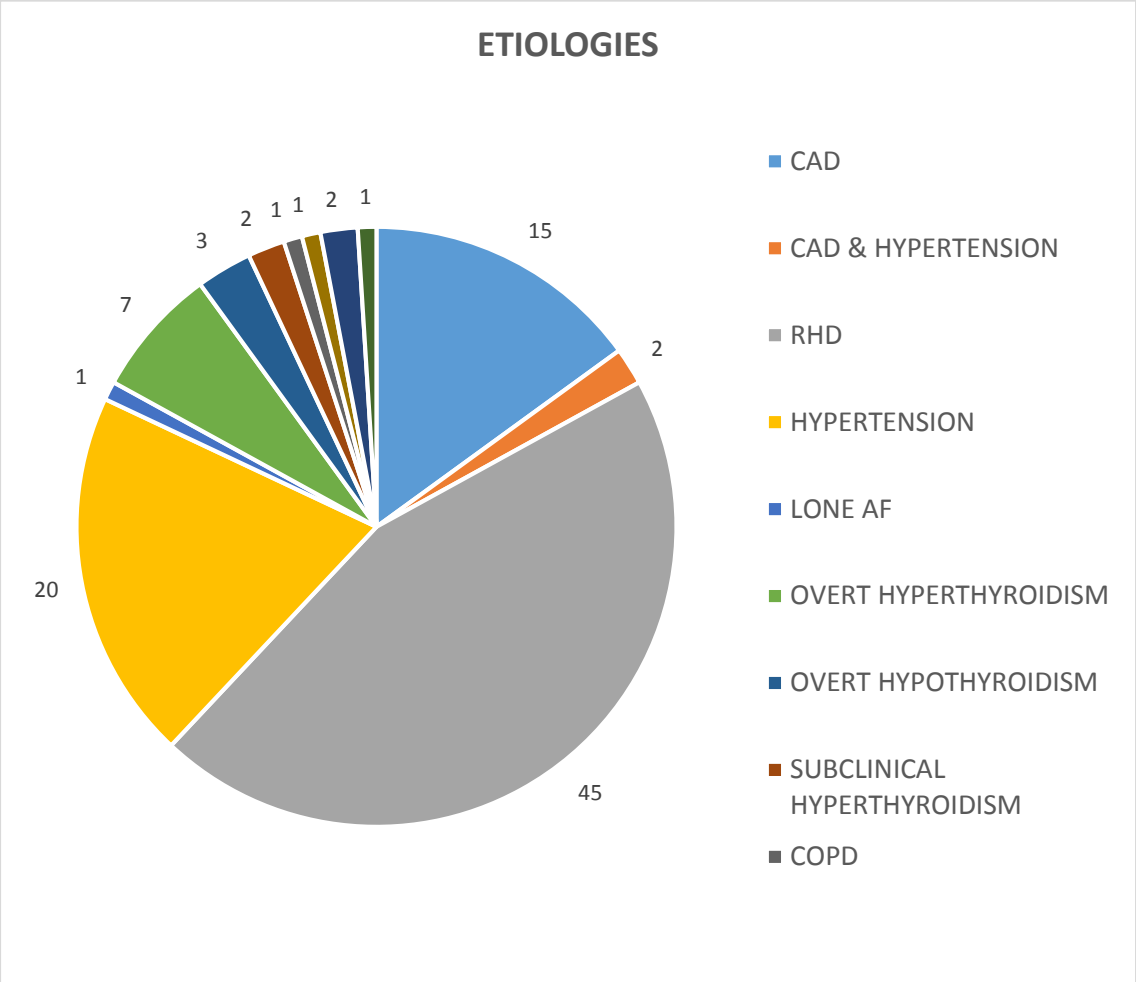


FIGURE 12: DISTRIBUTION ACCORDING TO ETIOLOGY

According to this study, the most common etiological factor that was associated with AF was Rheumatic Heart disease (45%). The next in line was hypertension (20%), closely followed by coronary artery disease (15%). The least common etiologies that were seen were cardiomyopathies, COPD, chronic pulmonary thromboembolic hypertension (CTEPH) and Lone AF, each of which contributed 1% of the total cases.

TABLE 11 : DISTRIBUTION ACCORDING TO HEART RATE:

HEART RATE GROUP	FREQUENCY
<100	35
100-140	53
>140	12
Total	**100**

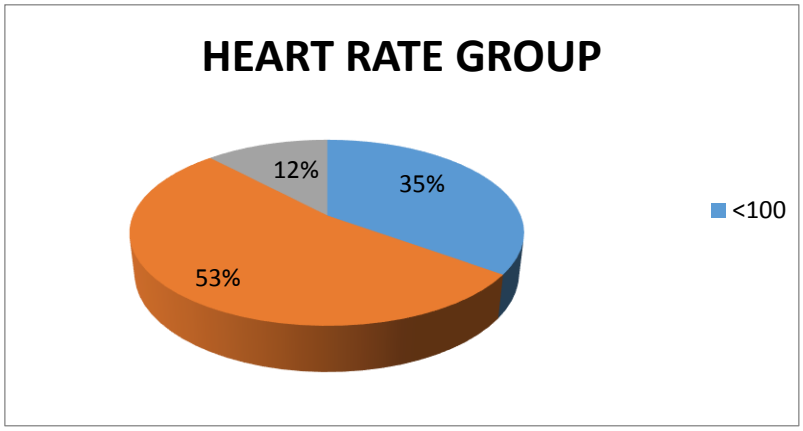


FIGURE 13: DISTRIBUTION ACCORDING TO HEART RATE

At presentation, majority had a heart rate between 100-140 (53%). Only 12% belonged to the heart rate group of less than 100.

TABLE 12 : COMPLICATION:

COMPLICATION	FREQUENCY
HEART FAILURE	32
THROMBOEMBOLISM	20

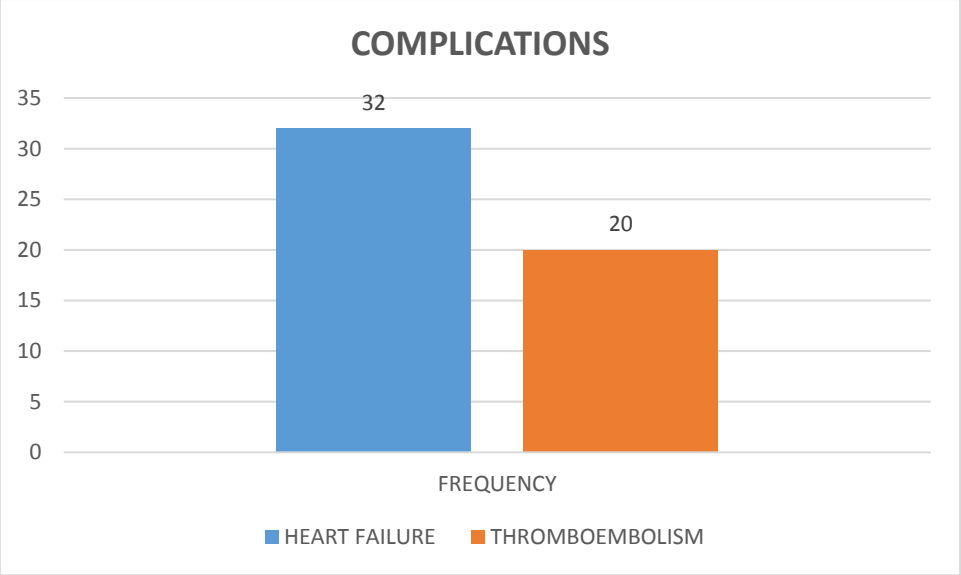


FIGURE 14: DISTRIBUTION ACCORDING TO COMPLICATIONS

The most common complication associated with atrial fibrillation in the present study was heart failure, seen in 32% of the patients. This was followed by thromboembolism(20%)

TABLE 13 : SEX DISTRIBUTION OF HEART FAILURE :

HEART FAILURE	FREQUENCY	PERCENTAGE
MALES	15	46%
FEMALES	17	54%
TOTAL	**32**	**100%**

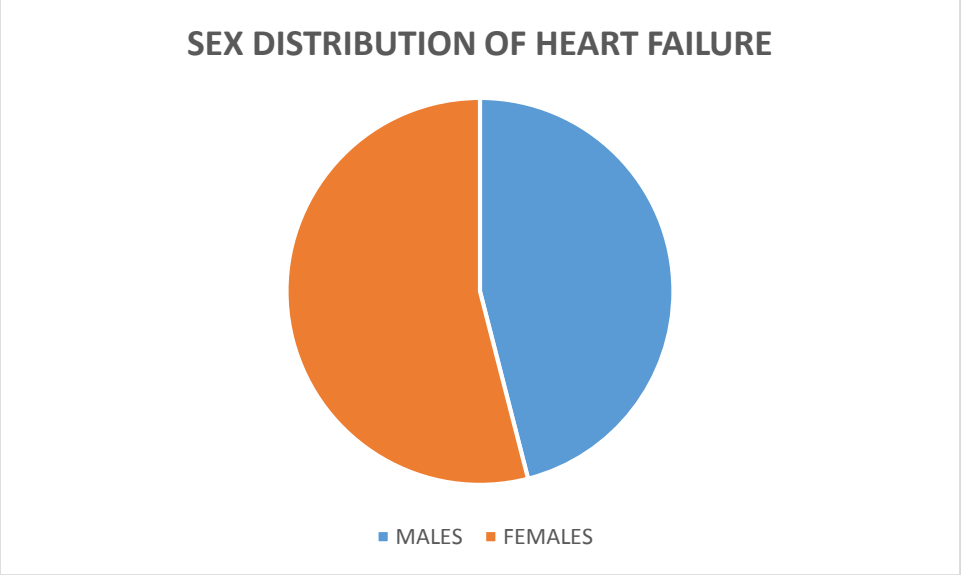


FIGURE 15: SEX DISTRIBUTION OF HEART FAILURE

Among the patients who developed heart failure , 54% were males and 46% were females.

Table 14 :ETIOLOGY DISTRIBUTION OF HEART FAILURE:

ETIOLOGY	FREQUENCY	PERCENTAGE
RHD	14	44%
CAD	12	38%
HYPERTENSION	3	9%
DCM	1	3%
COPD	1	3%
HYPOTHYROIDISM	1	3%
TOTAL	**32**	**100%**

Rheumatic heart disease contributed to the majority of heart failure cases (44%) followed by coronary artery disease (38%).

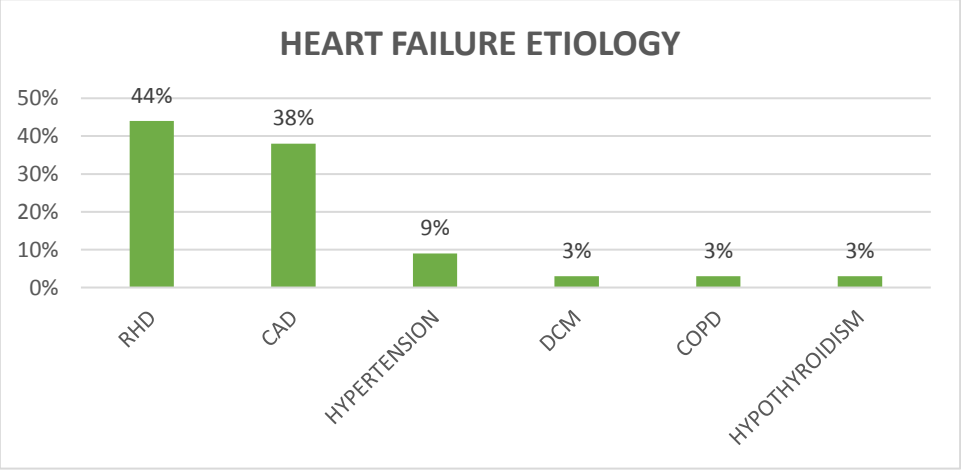


FIGURE 16 : ETIOLOGY DISTRIBUTION OF HEART FAILURE

TABLE 15 : PATTERN OF THROMBOEMBOLISM:

THROMBOEMBOLISM	FREQUENCY	PERCENTAGE
CVA	14	70%
PERIPHERAL EMBOLISM	6	30%
TOTAL	**20**	**100%**

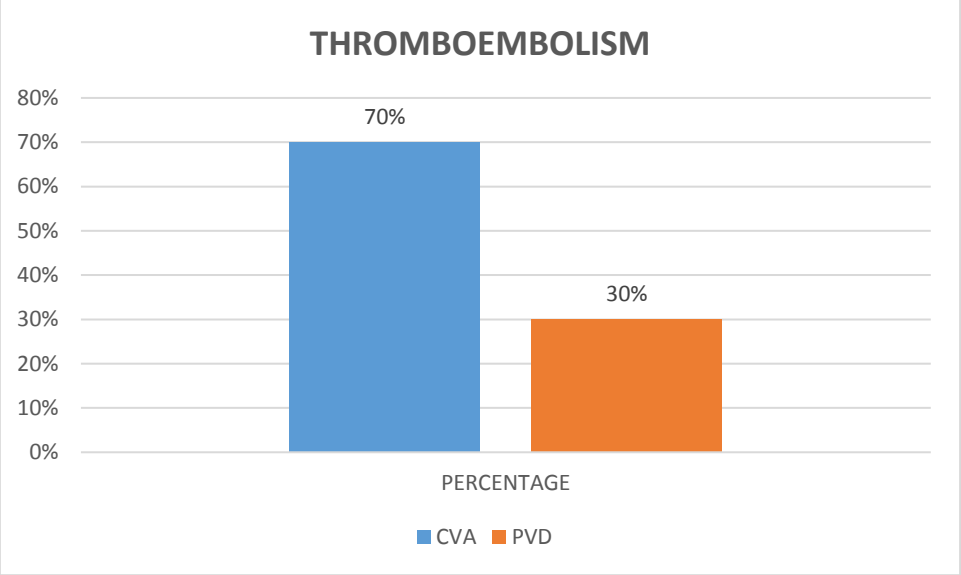


FIGURE 17 : PATTERN OF THROMBOEMBOLISM

Among the 20 patients who developed thromboembolism, 70% were due to CVA and the remaining 30% as a result of peripheral embolism.

TABLE 16: SEX DISTRIBUTION OF THROMBOEMBOLISM:

	MALES	FEMALES	TOTAL
CVA	7	7	14
PERIPHERAL EMBOLISM	0	6	6

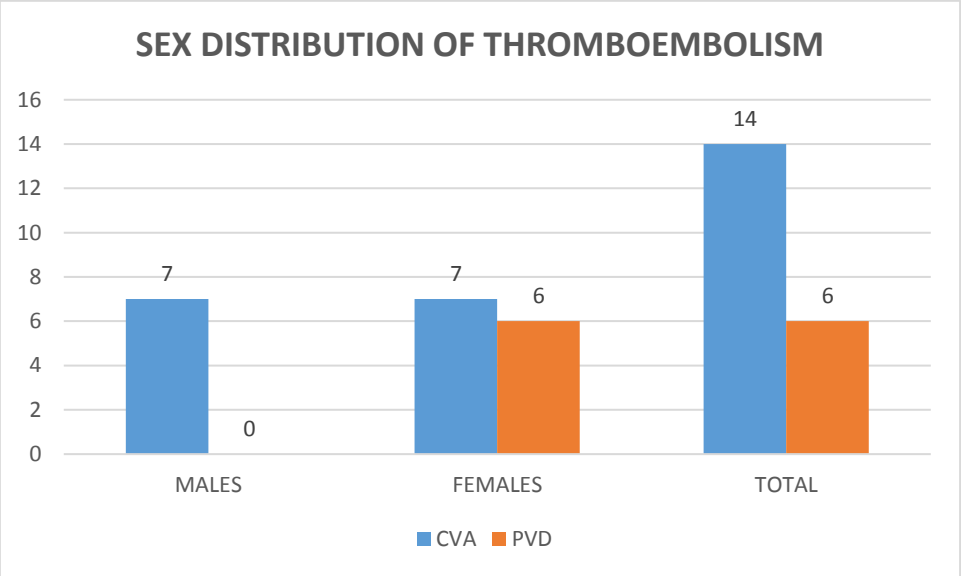


FIGURE 18 : SEX DISTRIBUTION OF THROMBOEMBOLISM

Among the 20 patients who developed thromboembolism, CVA was seen to have developed in an equal proportion in both males and females. On the contrary all the 6 patients who developed peripheral embolism were females.

TABLE 17 : ETIOLOGY DISTRIBUTION OF CVA:

ETIOLOGY	FREQUENCY	PERCENTAGE
CAD	6	43%
RHD	4	29%
HYPERTENSION	3	21%
DCM	1	7%
TOTAL	**14**	**100%**

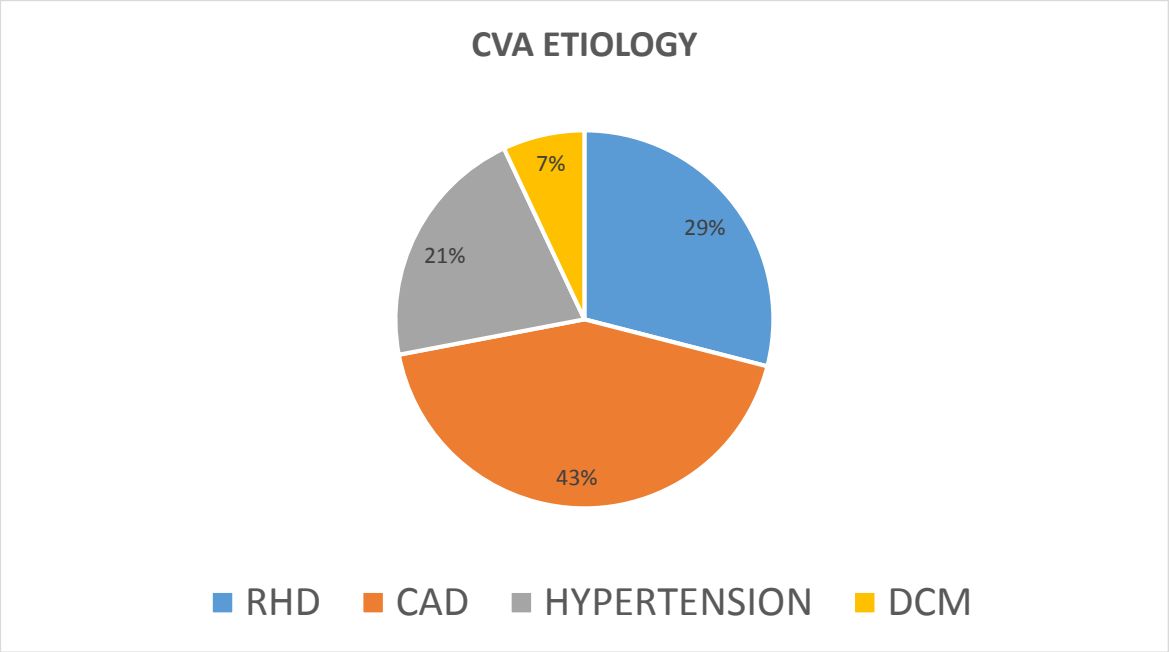


FIGURE 19 : ETIOLOGY DISTRIBUTION OF CVA

ETIOLOGY DISTRIBUTION OF PERIPHERAL EMBOLISM:

All the 6 patients who developed peripheral embolism were associated with rheumatic heart disease.

TABLE 18 : CHA2DS2VASC SCORE AMONG NON VALVULAR AF:

CHA2DS2VASc	Frequency	Percentage
0	13	23%
1	6	11%
2	11	20%
3	12	22%
4	6	11%
5	6	11%
6	1	2%
Total	**55**	**100%**

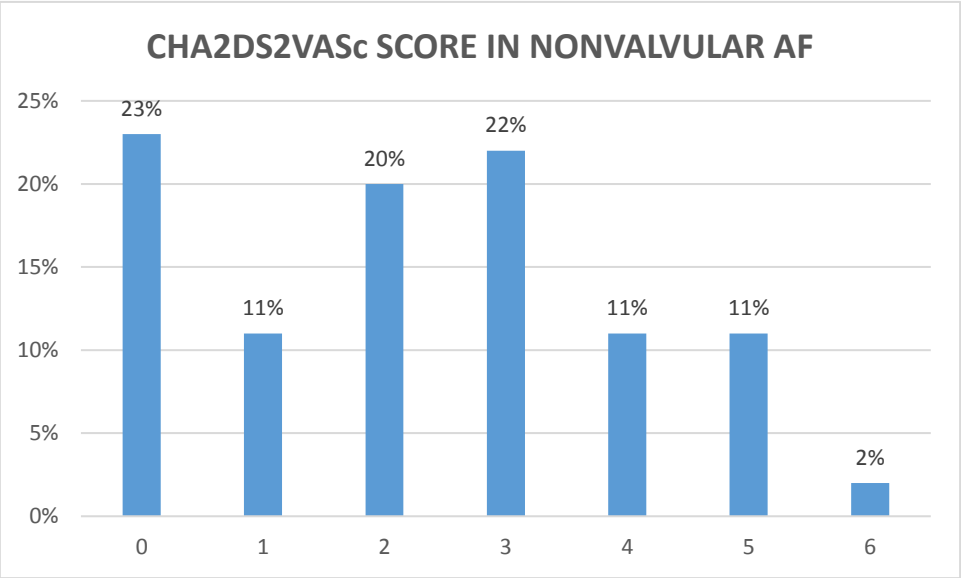


FIGURE 20: CHA2DS2VASc SCORE AMONG NON VALVULAR AF:

TABLE 19 :BLEEDING DUE TO ANTICOAGULATION

BLEEDING MANIFESTATION	FREQUENCY	PERCENT
INTRACEREBRAL HEMORRHAGE	3	33.3%
ECCHYMOSES	1	11.1%
EPISTAXIS	1	11.1%
GUM BLEED	1	11.1%
HEMATEMESIS	1	11.1%
CEREBELLAR HEMORRHAGE	1	11.1%
SUBDURAL HEMATOMA	1	11.1%
Total	**9**	**100.0%**

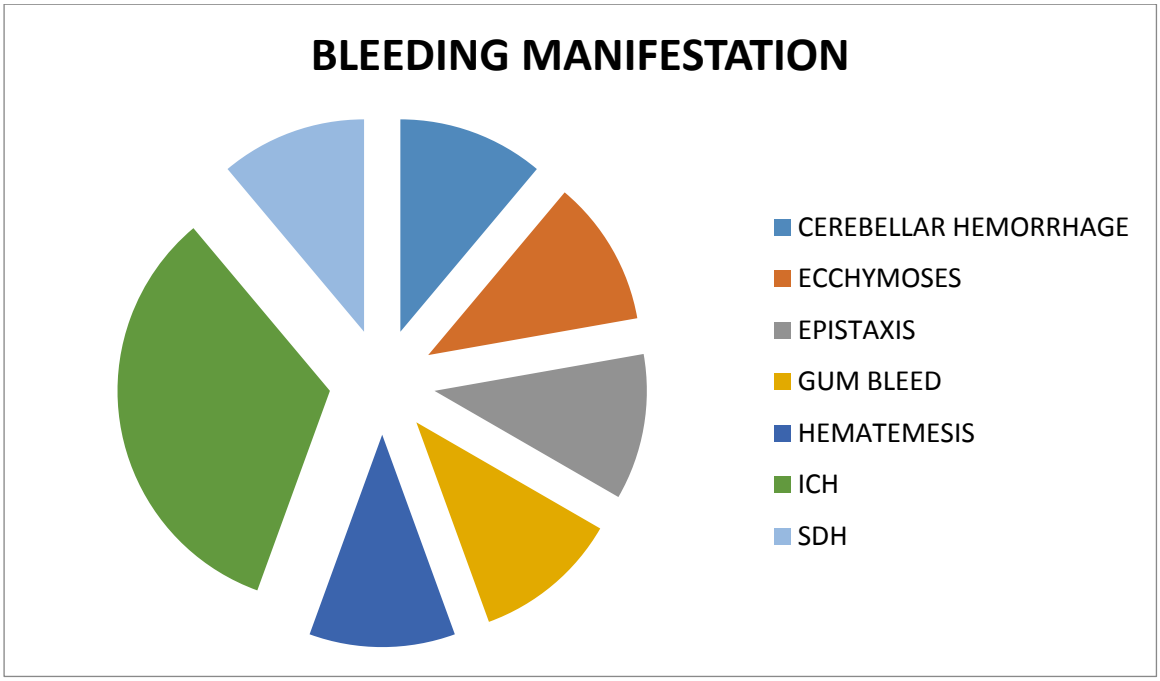


FIGURE 21: COMPLICATIONS DUE TO BLEEDING

Among the total number of 100 cases, 87 were anticoagulated ,of which 9 patients developed bleeding manifestations and illustrated above due to anticoagulation. 33.3 % of them suffered from intracerebral hemorrhage.

ECHOCARDIOGRAPHIC PARAMETERS:

TABLE 20 : VALVE LESION DISTRIBUTION AMONG RHD :

RHD	**Frequency**	**Percent**
MS	23	51%
MS.MR	13	30%
MS,AS,AR	6	13%
MS,MR,AS,AR	1	2%
S/P MVR	2	4%
TOTAL	**45**	**100%**

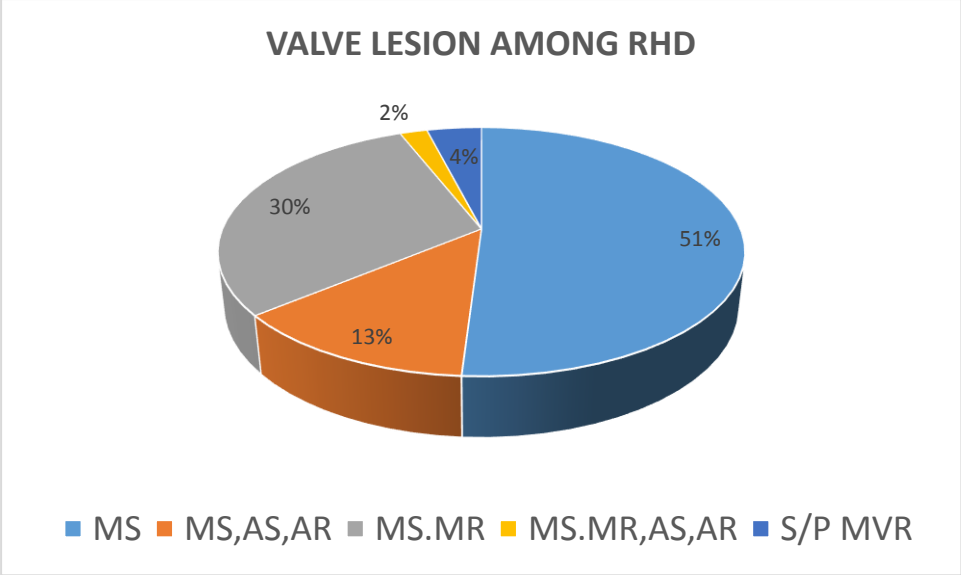


FIGURE 22 : VALVE LESION DISTRIBUTION AMONG RHD

Among the 45% of patients, mitral stenosis was the predominant valvular lesion (51%) , followed by MS with MR(30%). Aortic valve component was seen in 15% of the cases. 2 % were those who had undergone a mitral valve replacement.

TABLE 21 :PERCENTAGE OF LA AUTOCONTRAST:

LA AUTOCONTRAST	FREQUENCY
NO	87
YES	13
Total	**100**

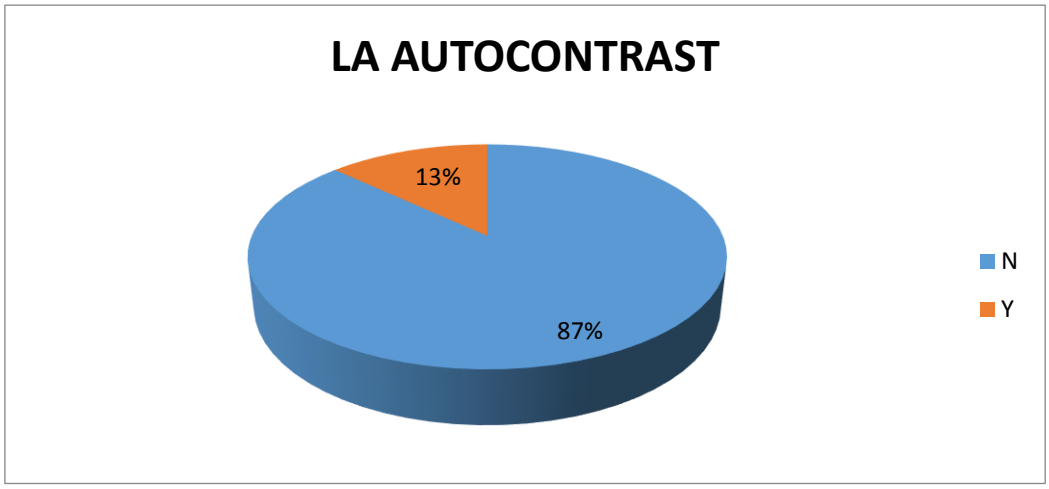


FIGURE 23 : PERCENTAGE OF LA AUTO CONTRAST

13% of the cases had a spontaneous echo contrast , whereas 87% did not.

LA AUTOCONTRAST AND CVA:

CVA was associated with 31% of those who had an LA auto contrast, while 11% of the patients with CVA did not have a spontaneous echo contrast.

LA AUTOCONTRAST AND PERIPHERAL EMBOLISM :

Among the patients who developed Peripheral embolism (6%) , none had a spontaneous echo contrast.

TABLE 22 : PERCENTAGE OF CLOT ON ECHO:

CLOT	Frequency
NO CLOT	85
LA CLOT	11
LV CLOT	3
PROSTHETIC VALVE THROMBOSIS	1
Total	**100**

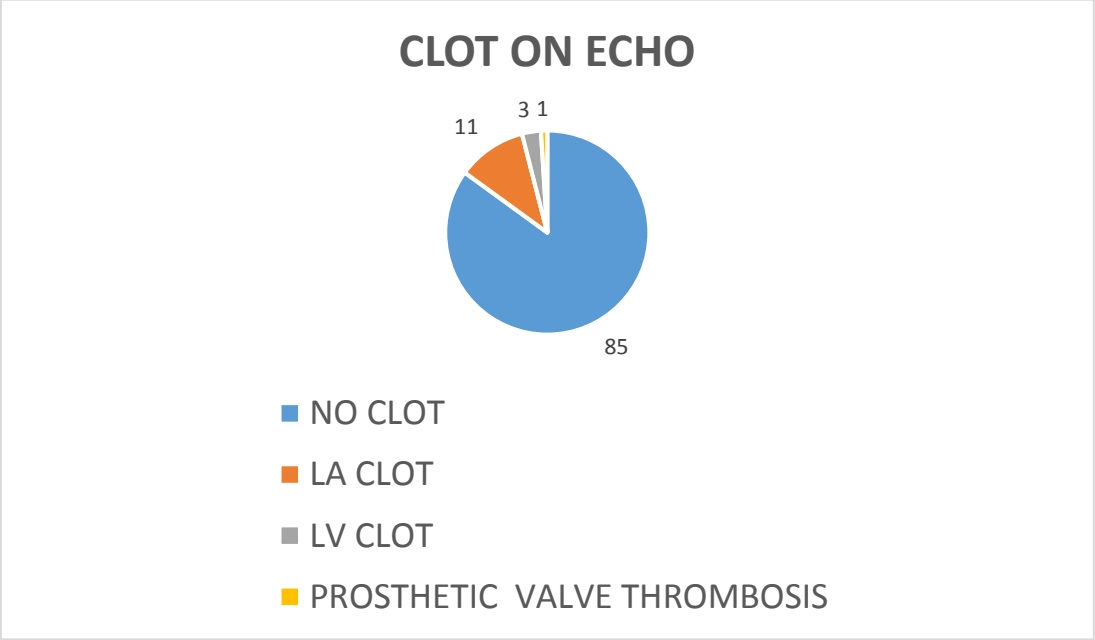


FIGURE 24 : PERCENTAGE OF CLOT ON ECHO

85% of the patients included in the study did not have a clot on transthoracic echocardiogram. The commonest clot that was observed was LA clot (11%) followed by LV clot(3%) and prosthetic valve thrombosis(1%).

TABLE 23 : HEART RATE AND CLOT ON ECHO :

			HR_RATE_GROUP			Total
			<100	100-140	>140	
CLOT	LA CLOT	Count	1	8	2	11
		% within HR_RATE_GROUP	2.9%	15.1%	16.7%	11.0%
	LV CLOT	Count	2	0	1	3
		% within HR_RATE_GROUP	5.7%	0.0%	8.3%	3.0%
	PR V CLOT	Count	0	0	1	1
		% within HR_RATE_GROUP	0.0%	0.0%	8.3%	1.0%
Total		Count	35	53	12	100
		% within HR_RATE_GROUP	100.0%	100.0%	100.0%	100.0%

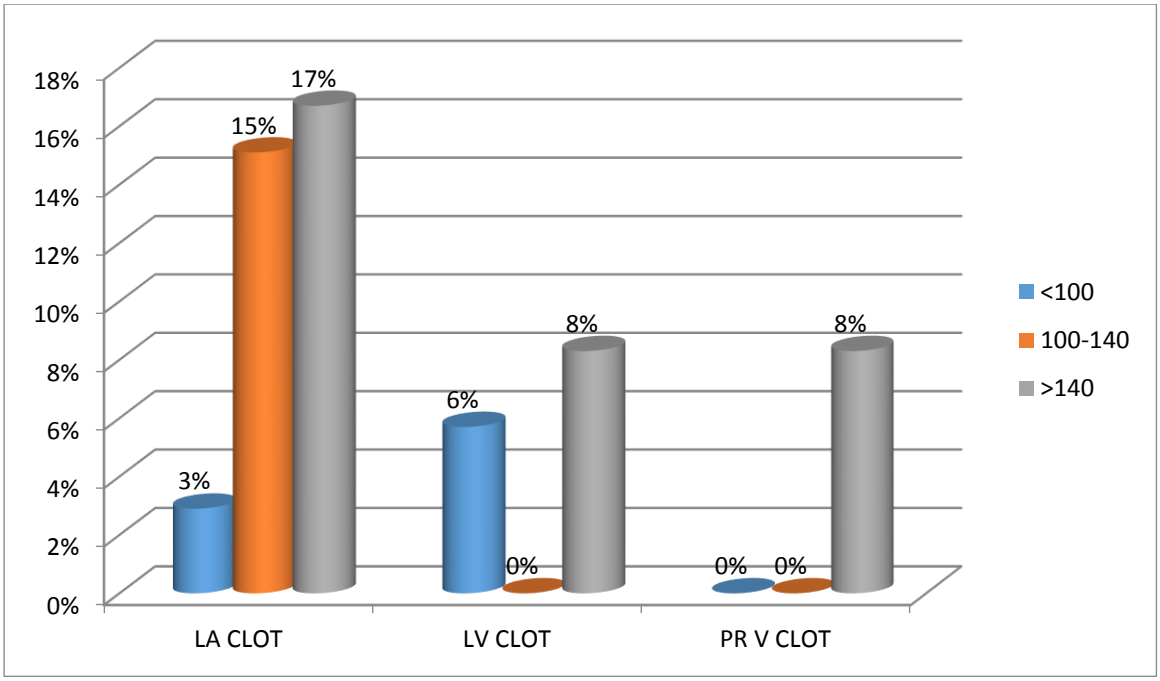


FIGURE 25: CLOT ON ECHO AND HEART RATE

33% of the patients who had a clot on echo, had presented with a rapid heart rate of more than 140/minute. Heart rate ranging between 100-140/min were associated with 15% of the patients with clot , whereas 9% with a heart rate less than 100/min had clot on echo.

TABLE 24 :CLOT ON ECHO AND CVA:

		CLOT				Total
		LA CLOT	LV CLOT	NO CLOT	PR V CLOT	
CVA	N	Count	7	0	78	1	86
		% within CLOT	63.6%	0.0%	91.7%	100.0%	86.0%
	Y	Count	4	3	7	0	14
		% within CLOT	36.4%	100.0%	82.3%	0.0%	14.0%
Total		Count	11	3	85	1	100
		% within CLOT	100.0%	100.0%	100.0%	100.0%	100.0%

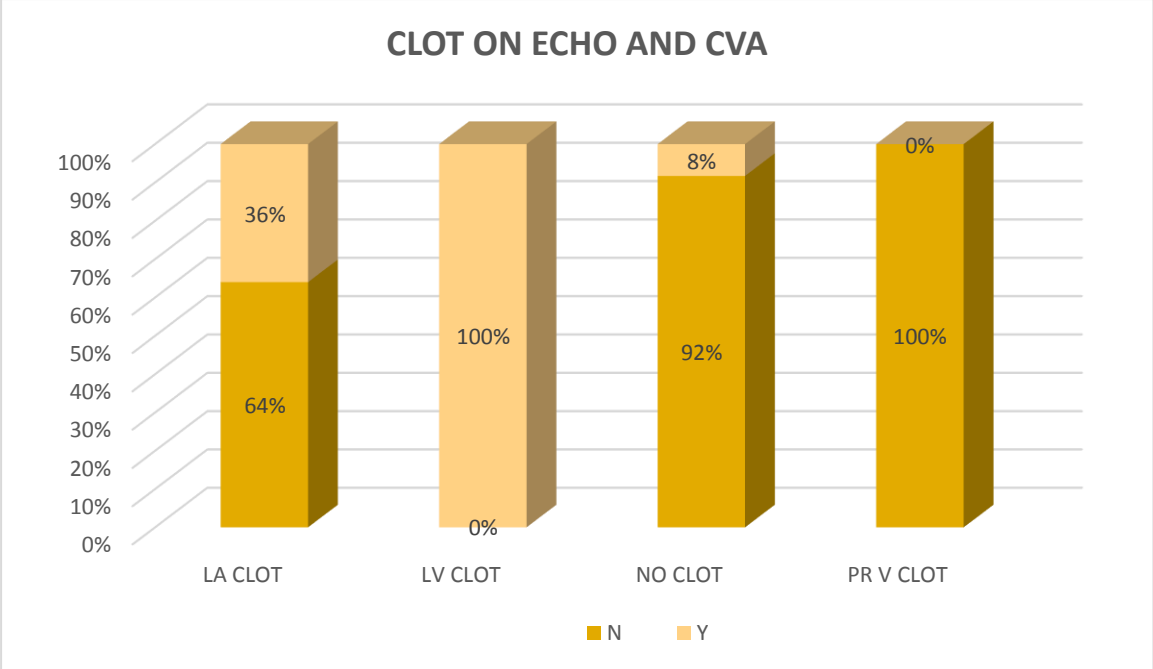


FIGURE26 : CLOT ON ECHO AND CVA

Among the 14 patients who developed CVA,7 patients had a clot on echo ,of which 4 had an LA clot and 3 patients had LV clot. The remaining 7 patients did not have a clot on echo.

Among the 15 patients who had a clot on echo,7 patients developed CVA whereas CVA was not observed in 8 patients.

TABLE 25 : CLOT ON ECHO AND PERIPHERAL EMBOLISM:

			CLOT				Total
			LA CLOT	LV CLOT	NO CLOT	PR V CLOT	
PVD	N	Count	6	3	84	1	94
		% within CLOT	54.5%	100.0%	98.8%	100.0%	94.0%
	Y	Count	5	0	1	0	6
		% within CLOT	45.5%	0.0%	1.2%	0.0%	6.0%
Total		Count	11	3	85	1	100
		% within CLOT	100.0%	100.0%	100.0%	100.0%	100.0%

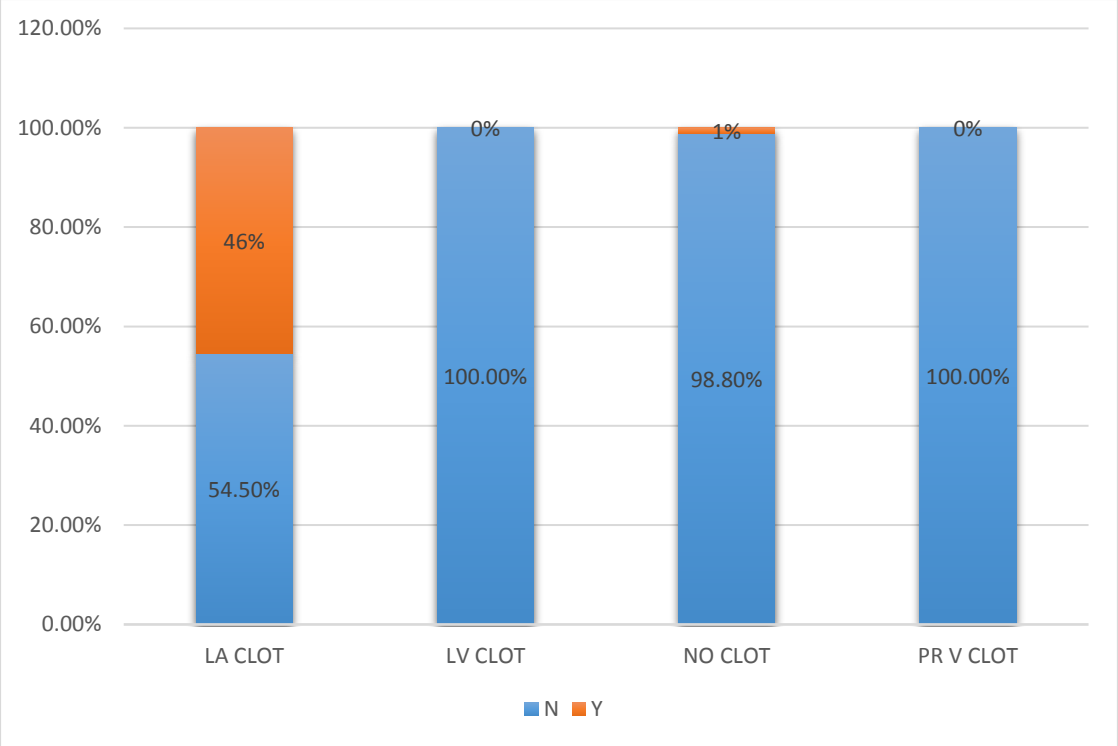


FIGURE 27 : CLOT ON ECHO AND PERIPHERAL EMBOLISM

Among the 6 patients who developed Peripheral embolism, 5 had LA clot and 1 patient had no clot on echo.

TABLE 26: DISTRIBUTION OF SEVERITY OF LV SYSTOLIC DYSFUNCTION

LVSD	FREQUENCY
NORMAL	85
MILD	5
MODERATE	4
SEVERE	11
Total	**100**

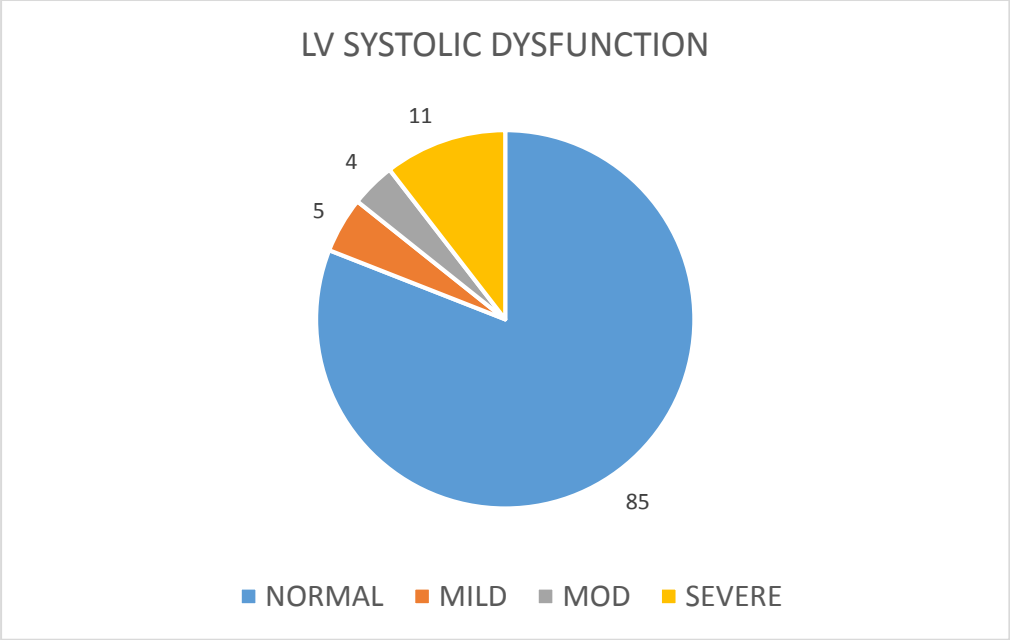


FIGURE28 : DISTRIBUTION OF LVSD

Out of the 100 patients, LVSD was seen in 19 patients , of which 11 had severe LVSD with an ejection fraction of 30%.

TABLE 27 : LEFT VENTRICULAR HYPERTROPHY

LVH	FREQUENCY
No	72
Yes	28
Total	**100**

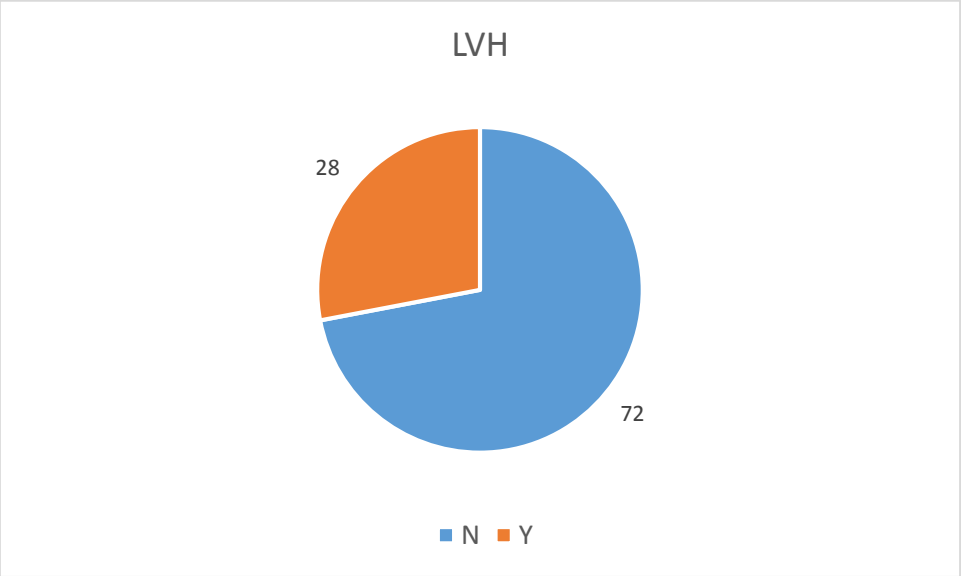


FIGURE 29 : PERCENTAGE OF LVH

Left ventricular hypertrophy was seen in 28% of the patients.

LA SIZE :

1. TABLE 28 :AGE DISTRIBUTION IN MALES:

			LA_size_male(cm)				Total
			<4.1	4.1-4.6	4.7-5.1	>5.1	
AGE_GROUP	UP TO 20 YEARS	Count	1	1	0	0	2
		%	11.1%	10.0%	0.0%	0.0%	4.0%
	21-40 YEARS	Count	5	0	2	4	11
		%	55.6%	0.0%	10.5%	33.3%	22.0%
	41-60 YEARS	Count	1	3	9	4	17
		%	11.1%	30.0%	47.4%	33.3%	34.0%
	61-80 YEARS	Count	2	6	8	4	20
		%	22.2%	60.0%	42.1%	33.3%	40.0%
Total		Count	9	10	19	12	50
		%	100.0%	100.0%	100.0%	100.0%	100.0%

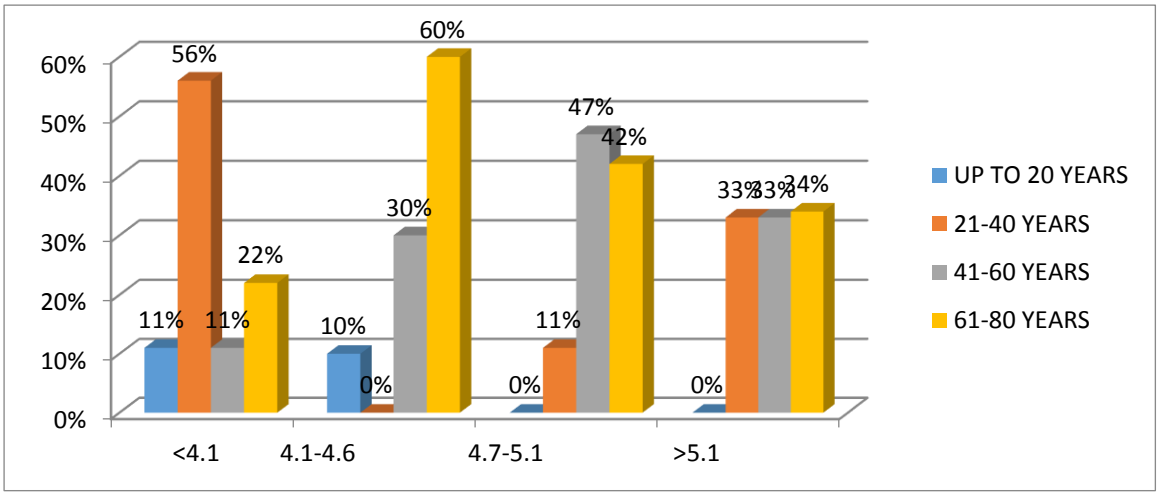


FIGURE 30– LA SIZE AND AGE DISTRIBUTION IN MALES

2. TABLE 29: AGE DISTRIBUTION IN FEMALES

			LA_size_Female(cm)				Total
			<3.9	3.9-4.2	4.3-4.6	>4.6	
Age_ Group	Up To 20 Years	Count	1	0	0	1	2
		%	16.7%	0.0%	0.0%	2.9%	4.0%
	21-40 Years	Count	5	3	2	11	21
		%	83.3%	75.0%	40.0%	31.4%	42.0%
	41-60 Years	Count	0	1	2	13	16
		%	0.0%	25.0%	40.0%	37.1%	32.0%
	61-80 Years	Count	0	0	1	10	11
		%	0.0%	0.0%	20.0%	28.6%	22.0%
Total		Count	6	4	5	35	50
		%	100.0%	100.0%	100.0%	100.0%	100.0%

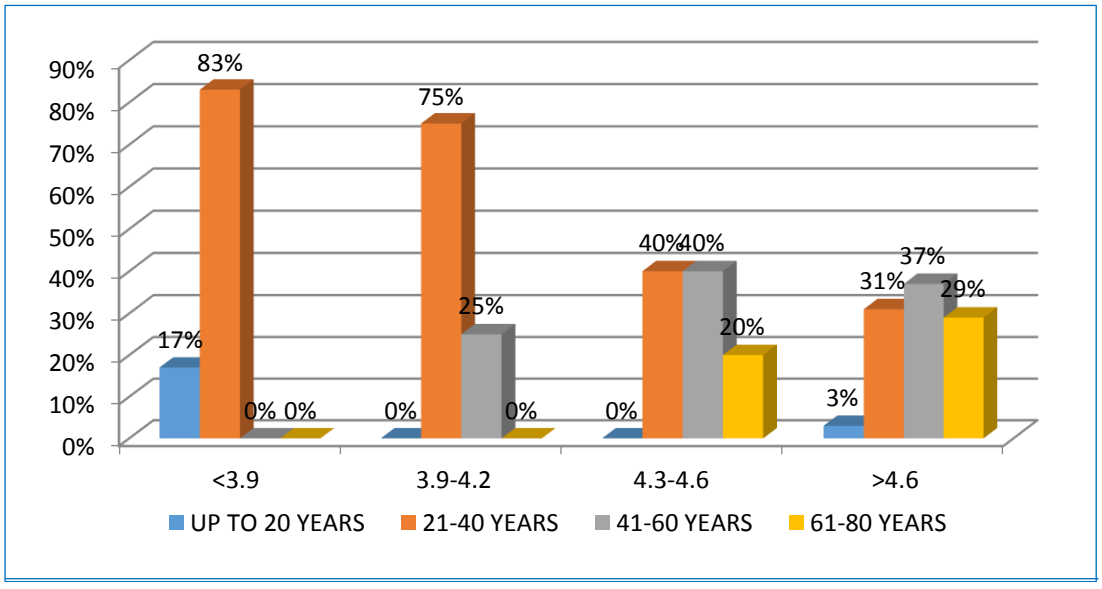


FIGURE 31: LA SIZE AGE DISTRIBUTION IN FEMALES

3. TABLE 30 : LA SIZE AND VALVULAR HEART DISEASE:

			LA_size_male(cm)			Total
			4.1-4.6	4.7-5.1	>5.1	
Lesion	MS	Count	1	5	1	7
		%	50.0%	50.0%	14.3%	36.8%
	MS, AS, AR	Count	1	4	1	6
		%	50.0%	40.0%	14.3%	31.6%
	MS,MR	Count	0	0	5	5
		%	0.0%	0.0%	71.4%	26.3%
	MS,MR, AS,AR	Count	0	1	0	1
		%	0.0%	10.0%	0.0%	5.3%
Total		Count	2	10	7	19
		%	100.0%	100.0%	100.0%	100.0%

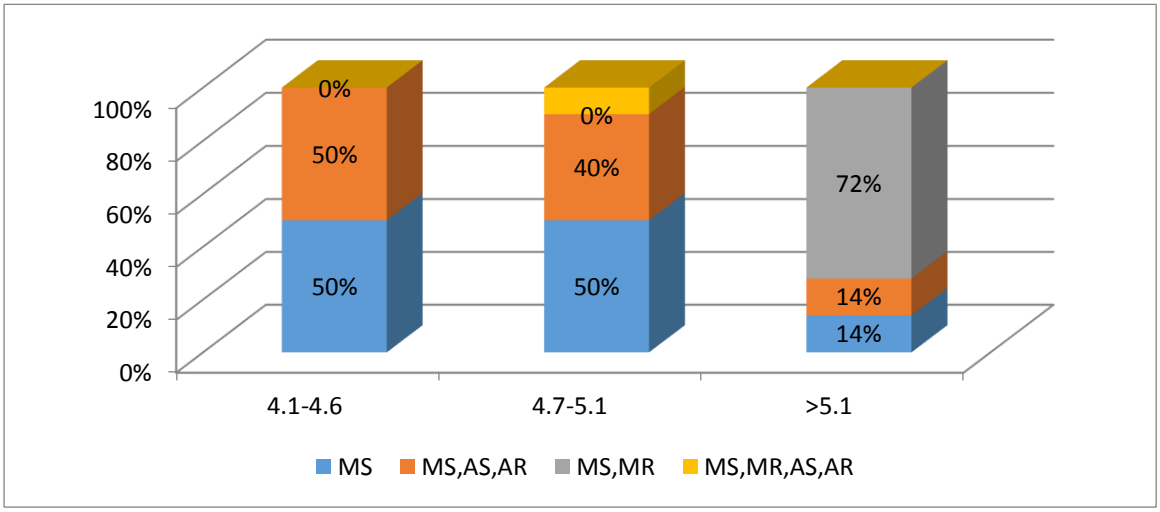


FIGURE 32: LA SIZE AND VALVULAR HEART DISEASE IN MALES

72% of patients with severe LA enlargement in males had MS, MR where as 14% had MS alone 14% had MS,AS,AR

4. TABLE 31 : LA SIZE AND VALVULAR HEART DISEASE IN FEMALES

			LA_size_Female(cm)			Total
			3.9-4.2	4.3-4.6	>4.6	
Lesion	MS	Count	2	2	10	14
		%	100.0%	66.7%	55.6%	60.9%
	MS, MR	Count	0	1	7	8
		%	0.0%	33.3%	38.9%	34.8%
	S/P MVR	Count	0	0	1	1
		%	0.0%	0.0%	5.6%	4.3%
Total		Count	2	3	18	23
		%	100.0%	100.0%	100.0%	100.0%

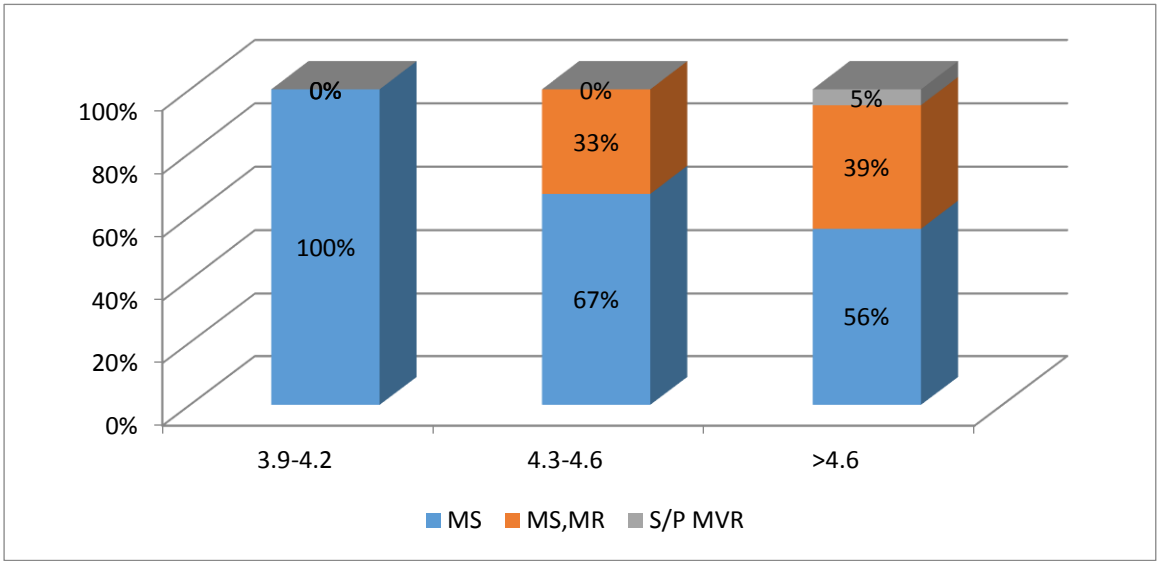


FIGURE 33: LA SIZE AND VALVULAR HEART DISEASE IN FEMALES

56% of females who had severe LA enlargement had pure MS while, 39% had MS, MR.

5. TABLE 32 : LA SIZE AND CLOT ON ECHO IN MALES :

			LA_size male(cm)				Total
			<4.1	4.1-4.6	4.7-5.1	>5.1	
Clot	LA Clot	Count	0	0	2	1	3
		%	0.0%	0.0%	10.5%	8.3%	6.0%
	LV Clot	Count	1	0	0	0	1
		%	11.1%	0.0%	0.0%	0.0%	2.0%
Total			1	0	2	1	4

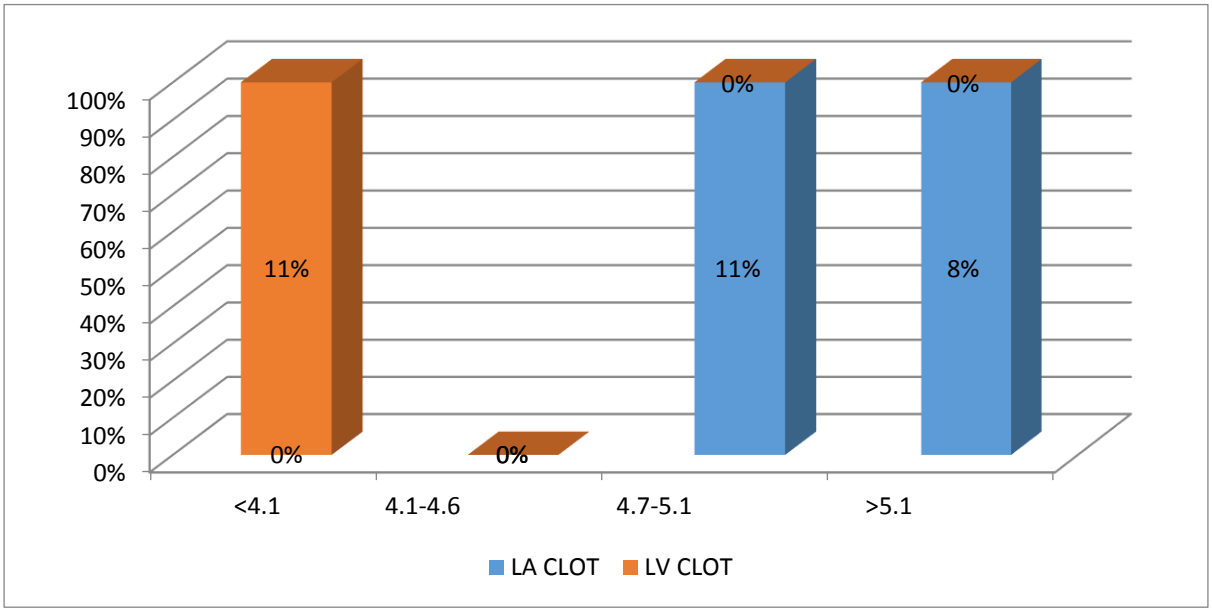


FIGURE 34 : LA SIZE AND CLOT ON ECHO IN MALES

All male patients who had LA clot were having LA enlargement.

6. TABLE 33 : LA SIZE AND CLOT ON ECHO IN FEMALES

		LA_size_Female(cm)				Total
		<3.9	3.9-4.2	4.3-4.6	>4.6	
CLOT	LA CLOT	Count	0	0	0	8	8
		%	0.0%	0.0%	0.0%	22.9%	16.0%
	LV CLOT	Count	0	0	0	2	2
		%	0.0%	0.0%	0.0%	5.7%	4.0%
	PR V CLOT	Count	0	0	0	1	1
		%	0.0%	0.0%	0.0%	2.9%	2.0%
Total		Count	0	0	0	11	11

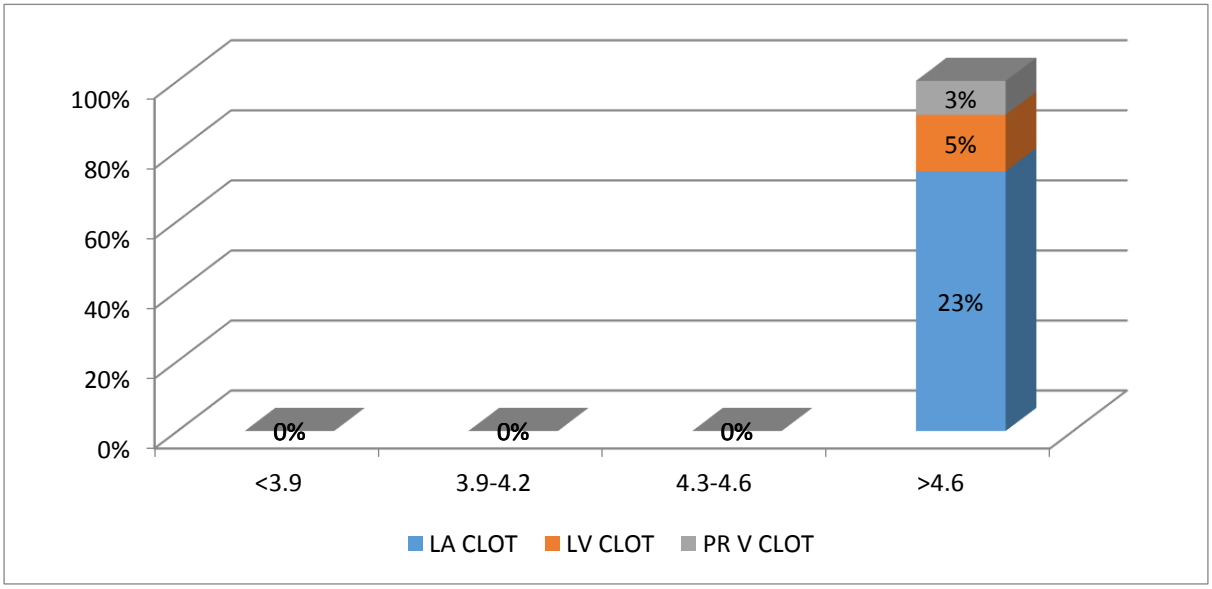


FIGURE35 : LA SIZE AND CLOT ON ECHO IN FEMALES

All female patients who had clot on echo were seen to have severe LA enlargement.

7. TABLE 34: LA SIZE AND CVA IN MALES:

			LA_size_male			Total
			<4.1	4.1-4.6	4.7-5.1	>5.1	
CVA	N	Count	8	10	16	9	43
		%	88.9%	100.0%	84.2%	75.0%	86.0%
	Y	Count	1	0	3	3	7
		%	11.1%	0.0%	15.8%	25.0%	14.0%
Total		Count	9	10	19	12	50
		%	100.0%	100.0%	100.0%	100.0%	100.0%

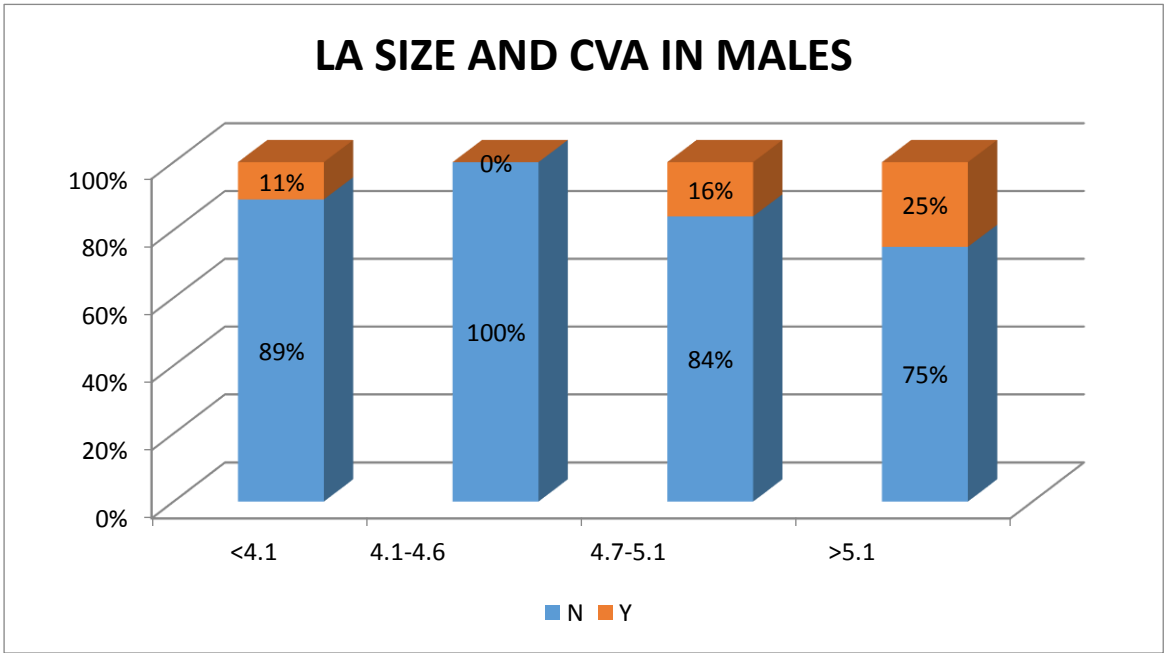


FIGURE 36 : LA SIZE AND CVA IN MALES

Among the male patients with LA enlargement, 25% with severe LA enlargement had CVAand 16% with moderate LA enlargement had CVA.

8. TABLE 35: LA SIZE AND CVA IN FEMALES:

			LA_size_male_Female			Total
			<3.9	3.9-4.2	4.3-4.6	>4.6	
CVA	N	Count	6	4	3	25	38
		%	100.0%	100.0%	60.0%	71.4%	76.0%
	Y	Count	0	0	2	10	12
		%	0.0%	0.0%	40.0%	28.6%	24.0%
Total		Count	6	4	5	35	50
		%	100.0%	100.0%	100.0%	100.0%	100.0%

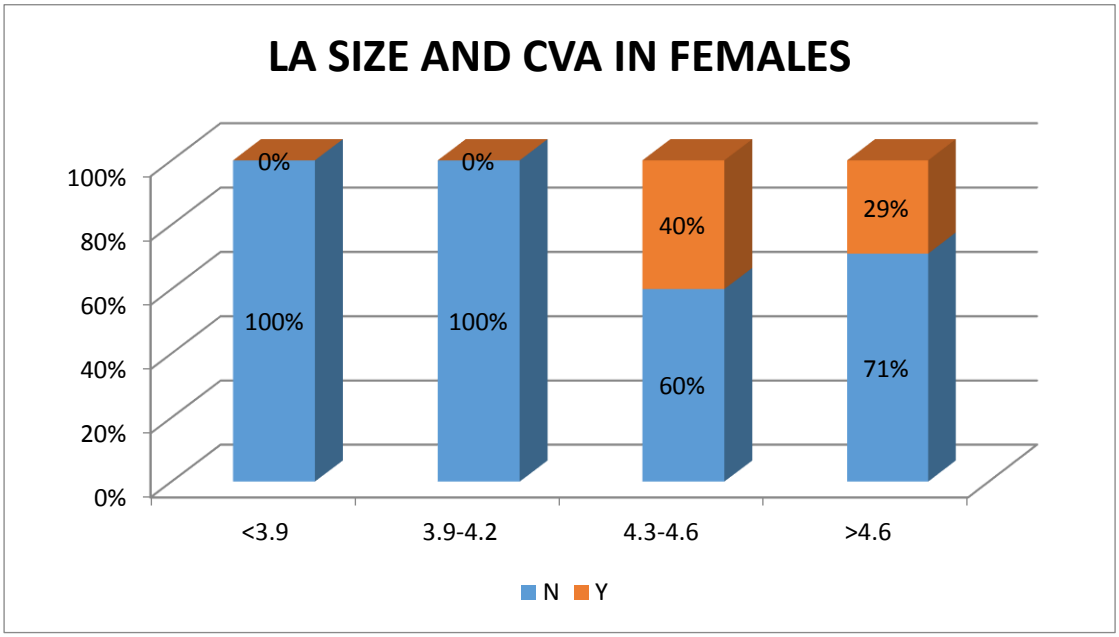


FIGURE 37– LA SIZE AND CVA IN FEMALES

Among the female patients with LA enlargement, 29% with severe enlargement had CVA and 40% with moderate enlargement had CVA.

DISCUSSION

The present study was undertaken to analyse the etiological, clinical and echocardiographic profile of atrial fibrillation and its complications.

AGE:

The mean age of AF in the present study was 49 years. Benjamin.O et al[44] reported mean age as 58 in their study. AFFIRM study[45] has reported mean age as 70 years. The mean age is higher in their studies as most of the population was above 50 years of age in their study group, which differed from the present study.

SEX:

Males and females were seen to be affected equally in our study. According to AFFIRM study, incidence in males was 61% Framingham heart study[46] reported a higher incidence in males.

ETIOLOGY:

RHD:

In our study out of 100 cases, 45 had rheumatic heart disease as the etiology. Bernard L.J et al[47] concluded in their study that 70% of their population had RHD as the etiology. AFFIRM study reports very low incidence of RHD in their study. Studies that were done in tropical countries all concur with a higher incidence of atrial fibrillation in RHD as compared to the Western population.

HYPERTENSION:

The next common cause of AF in our study was hypertension seen in 20% of the cases. According to the Framingham study[46] , half of the cases were accounted by hypertension. In the AFFIRM study[45] , hypertension was present in 71%. The association with hypertension is lesser in our study than the above trials.

CORONARY ARTERY DISEASE:

CAD was present in 15% of the cases in our study. In the AFFIRM[45] study, CAD was present in 38% of the cases. Sameul Levy [48] et al reported 16.6% of CAD as the underlying cause

OTHER ETIOLOGIES:

Thyroid dysfunction was seen in 12 cases of which 7 had overt hyperthyroidism, 2 had subclinical hyperthyroidism and 3 had overt hypothyroidism. Auer J et al[49] reported that AF occurred in 13.8% with overt hyperthyroidism and 12.7% were seen with subclinical hyperthyroidism. Barbisan et al[50] in their study noted that out of 72 patients with atrial fibrillation, 16.6% had thyroid dysfunction out of which 5.6% had hypothyroidism.

SYMPTOM ANALYSIS:

Dyspnea was the most common symptom seen in 81% of the patients followed by palpitation in 44%. Dyspnea was reported by Tischer et al[51] in 62% of patients and palpitation in 33%. Flaker et al[52] noted that 78% had dyspnea and 11% had chest pain.

COMPLICATIONS:

Heart failure was seen in 32% of patients and thromboembolism was seen in 20% in our study. AFFIRM[45] trial suggests that 32% of their patients had left ventricular dysfunction. 14 patients in our study had cerebral embolism and 6 had peripheral embolism. CVA was predominant in Coronary artery disease (43% of CVA was due to CAD) with a higher CHA2DS2Vasc score, while peripheral embolism was predominant in females of rheumatic heart disease patients in our study.

Bleeding manifestations as a complication of anticoagulation was noted in 9% of the cases in our study. All of them had an INR>4.

ECHOCARDIOGRAPHIC FEATURES:

VALVE LESION:

Among the 45% of patients, mitral stenosis was the predominant valvular lesion (51%) , followed by MS with MR(30%). Aortic valve component was seen in 15% of the cases. SK Sharma et al observed in their study that Pure mitral stenosis was seen 48% of cases . Predominant mitral regurgitation 35% of cases. The AFFIRM Investigators found that mitral regurgitation was present in 15% of their cases.

LA AUTO CONTRAST:

13 patients with AF had spontaneous echo contrast of which 4 were associated with CVA in our study.

CLOT on ECHOCARDIOGRAPHY:

11 patients had an LA clot, 3 had LV clot and 1 patient had prosthetic valve thrombosis. 87% did not have clot on echo. 33% of the patients who had a clot on echo, had presented with a rapid heart rate of more than 140/minute.

CLOT ON ECHO AND CVA:

Among the 14 patients who developed CVA while 7 patients ,4 had an LA clot and 3 patients had LVclot. The remaining 7 patients did not have a clot on echo. Among the 15 patients who had a clot on echo,7 patients developed CVA whereas CVA was not observed in 8 patients. Among the 6 patients who developed PVD, 5 had LA clot and 1 patient had no clot on echo. Studies by Wood e t al and Deverall P et al observed that systemic embolization occurs primarily in the presence of atrial fibrillation.

LA SIZE :

72% of patients with severe LA enlargement in males had MS, MR where as 14% had MS alone 14% had MS,AS,AR.56% of females who had severe LA enlargement had pure MS while, 39% had MS,MR.

All male patients who had LA clot were having LA enlargement. All female patients who had clot on echo were seen to have severe LA enlargement. .Mahmood ul Hassan et al[57] noted in their study that the frequency of left atrial and appendage clots on trans-oesophageal echocardiography in patients with

severe mitral stenosis is common and more frequent in patients with AF and LA size > or = 45 mm.

Among the male patients with LA enlargement, 25% with severe LA enlargement had CVA and 16% with moderate LA enlargement had CVA.Among the female patients with LA enlargement, 29% with severe enlargement had CVA and 40% with moderate enlargement had CVA. JieXue et al[56] noted in their study of 313 patients that LA size is an independent predictor of total recurrent ischemic stroke and the composite of recurrent cardioembolic or cryptogenic stroke.

LEFT VENTRICULAR SYSTOLIC DYSFUNCTION:

LVSD was seen in 19 patients , out of which 58% had severe LVSD with ejection fraction of 30%. AFFIRM[45] trial suggests that 32% of their patients had left ventricular dysfunction. LV systolic dysfunction (ejection fraction <50%) occurred in 25% of cases noted by Teicholz et al[55]

LEFT VENTRICULAR HYPERTROPHY:

Left ventricular hypertrophy was seen in 28% of the patients.Teicholz et al[55] observed that LAE and LVH with normal EF are commoner in hypertensive pts with suggesting diastolic dysfunction.

CONCLUSION

According to this study,

1. The mean age of atrial fibrillation was 49 years,males and females being equally affected.
2. Dyspnea was the most common presenting symptom followed by palpitation,
3. RHD was the most common etiological factor followed by hypertension and CAD.
4. Predominant valve affected is the mitral valve both in isolation and in combination.
5. Heart failure was the most common complication followed by thromboembolism
6. Stroke was more common than peripheral embolism
7. Females of rheumatic heart disease had the highest frequency of peripheral embolism.
8. The presence of clot on echo increases the risk of thromboembolism
9. Higher heart rate > 140/min is a risk factor for clot formation.
10. LA enlargement had a higher incidence of clot on echocardiography
11. LA enlargement had a higher incidence of ischemic stroke.

Meticulous evaluation of risk factors and prompt treatment will reduce the complications, morbidity and mortality.

LIMITATIONS

1. Sample size was small due to time constraint
2. The study was conducted in patients admitted at a single tertiary care center.
3. This was a cross sectional study and follow up details were not evaluated.
4. Transesophageal echo was not done for better detection of clot.

REFERENCES

1. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics—2012 update: a report from the American Heart Association. Circulation. 2012;125:e2–e220.

2. January CT, Wann LS, Alpert JS, et al. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. J AmCollCardiol. 2014;64:e1–e76

3. Gregory F. Michaud, William G. Stevenson Supraventricular Tachyarrhythmias Harrison's principles of internal medicine 1485-1488

4. Paulus Kirchhof*Stefano Benussi*1 Dipak Kotechaet al2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS European Heart Journaldoi:10.1093/eurheartj/ehw210

5. Narayan SM, Krummen DE, Shivkumar K, et al. Treatment of atrial fibrillation by the ablation of localized sources: CONFIRM (Conventional Ablation for Atrial Fibrillation With or Without Focal Impulse and Rotor Modulation) trial. J Am CollCardiol. 2012;60:628–636.

6. Gudbjartsson DF et al. Variants conferring risk of atrial fibrillation on chromosome 4q25. Nature 2007;448:353–357.

7. Lubitz SA et al. Novel genetic markers associate with atrial fibrillation risk in Europeans and Japanese. J Am CollCardiol 2014;63:1200–1210.

8. Wakili R, Voigt N, Kaab S, et al. Recent advances in the molecular pathophysiology of atrial fibrillation. J Clin Invest. 2011;121:2955–2968.

9. Kannel WB, Abbott RD, Savage DD, McNamara PM. Epidemiologic features of chronic atrial fibrillation: the Framingham study. N Engl J Med 1982; 306:1018.

10. Krahn AD, Manfreda J, Tate RB, et al. The natural history of atrial fibrillation: incidence, risk factors, and prognosis in the Manitoba Follow-Up Study. Am J Med 1995; 98:476.

11. Liberthson RR, Salisbury KW, Hutter AM Jr, DeSanctis RW. Atrial tachyarrhythmias in acute myocardial infarction. Am J Med 1976; 60:956.

12. Crenshaw BS, Ward SR, Granger CB, et al. Atrial fibrillation in the setting of acute myocardial infarction: the GUSTO-I experience. Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries. J Am CollCardiol 1997; 30:406.

13. Diker E, Aydogdu S, Ozdemir M, et al. Prevalence and predictors of atrial fibrillation in rheumatic valvular heart disease. Am J Cardiol 1996; 77:96.

14. Santhanakrishnan R, Wang N, Larson MG, et al. Atrial Fibrillation Begets Heart Failure and Vice Versa: Temporal Associations and Differences in Preserved Versus Reduced Ejection Fraction. Circulation 2016; 133:484.

15. Glancy DL, O'Brien KP, Gold HK, Epstein SE. Atrial fibrillation in patients with idiopathic hypertrophic subaortic stenosis. Br Heart J 1970; 32:652.

16. Tikoff G, Schmidt AM, Hecht HH. Atrial fibrillation in atrial septal defect. Arch Intern Med 1968; 121:402.

17. Hald EM, Enga KF, Løchen ML, et al. Venous thromboembolism increases the risk of atrial fibrillation: the Tromso study. J Am Heart Assoc 2014; 3:e000483.

18. Kanagala R, Murali NS, Friedman PA, et al. Obstructive sleep apnea and the recurrence of atrial fibrillation. Circulation 2003; 107:2589.

19. Mahajan R, Lau DH, Brooks AG, et al. Electrophysiological, Electroanatomical, and Structural Remodeling of the Atria as Consequences of Sustained Obesity. J Am CollCardiol 2015; 66:1.

20. Wang TJ, Parise H, Levy D, et al. Obesity and the risk of new-onset atrial fibrillation. JAMA 2004; 292:2471.

21. Devereux RB, Roman MJ, Paranicas M, et al. Impact of diabetes on cardiac structure and function: the strong heart study. Circulation 2000; 101:2271.

22. Watanabe H, Watanabe T, Sasaki S, et al. Close bidirectional relationship between chronic kidney disease and atrial fibrillation: the Niigata preventive medicine study. Am Heart J 2009; 158:629.

23. Lubitz SA, Yin X, Rienstra M, et al. Long-term outcomes of secondary atrial fibrillation in the community: the Framingham Heart Study. Circulation 2015; 131:1648.

24. Maisel WH, Rawn JD, Stevenson WG. Atrial fibrillation after cardiac surgery. Ann Intern Med 2001; 135:1061.

25. Pires LA, Wagshal AB, Lancey R, Huang SK. Arrhythmias and conduction disturbances after coronary artery bypass graft surgery: epidemiology, management, and prognosis. Am Heart J 1995; 129:799.

26. Pavri BB, O'Nunain SS, Newell JB, et al. Prevalence and prognostic significance of atrial arrhythmias after orthotopic cardiac transplantation. J Am CollCardiol 1995; 25:1673.

27. Ahmari SA, Bunch TJ, Chandra A, et al. Prevalence, pathophysiology, and clinical significance of post-heart transplant atrial fibrillation and atrial flutter. J Heart Lung Transplant 2006; 25:53.

28. Gialdini G, Nearing K, Bhave PD, et al. Perioperative atrial fibrillation and the long-term risk of ischemic stroke. JAMA 2014; 312:616.

29. Bhave PD, Goldman LE, Vittinghoff E, et al. Incidence, predictors, and outcomes associated with postoperative atrial fibrillation after major noncardiac surgery. Am Heart J 2012; 164:918.

30. Frost L, Vestergaard P, Mosekilde L. Hyperthyroidism and risk of atrial fibrillation or flutter: a population-based study. Arch Intern Med 2004; 164:1675.

31. Auer J, Scheibner P, Mische T, et al. Subclinical hyperthyroidism as a risk factor for atrial fibrillation. Am Heart J 2001; 142:838.

32. Wynn GJ, Todd DM,Webber M, Bonnett L, McShane J, Kirchhof P, Gupta D. The European Heart Rhythm Association symptom classification for atrial fibrillation: validation and improvement through a simple modification. Europace 2014;16: 965–972.

33. Krahn AD, Klein GJ, Kerr CR, et al. How useful is thyroid function testing in patients with recent-onset atrial fibrillation? The Canadian Registry of Atrial Fibrillation Investigators. Arch Intern Med 1996; 156:2221.

34. Lip GY, Nieuwlaat R, Pisters R, Lane DA, Crijns HJ. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: the euro heart survey on atrial fibrillation. Chest 2010;137:263–272.

35. Senoo K, Proietti M, Lane DA, et al Evaluation of the HAS-BLED, ATRIA and ORBIT bleeding risk scores in atrial fibrillation patients on warfarin. Am J Med. 2016;129:600–607.

36. Vamos M, Erath JW, Hohnloser SH. Digoxin-associated mortality: a systematic review and meta-analysis of the literature. Eur Heart J. 2015;36:1831–1838.

37. Allen LaPointe NM, Dai D, Thomas L, et al. Antiarrhythmic drug use in patients<65 years with atrial fibrillation and without structural heart disease. Am J Cardiol.2015;115:316–322.

38. Segal JB, McNamara RL, Miller MR, Kim N, Goodman SN, Powe NR, Robinson K,Yu D, Bass EB. The evidence regarding the drugs used for ventricular rate control. In. J Fam Practice 2000; 49:47–59.

39. Schreck DM, Rivera AR, Tricarico VJ. Emergency management of atrial fibrillation and flutter: intravenous diltiazem versus intravenous digoxin. Ann Emerg Med1997;29:135–140.

40. Ziff OJ, Lane DA, Samra M, Griffith M, Kirchhof P, Lip GY, Steeds RP, Townend J,Kotecha D. Safety and efficacy of digoxin: systematic

review and meta-analysis of observational and controlled trial data. BMJ 2015;351:h4451.

41. Jonathan P Piccini, Laurent Fauchier Rhythm control in atrial fibrillation Lancet 2016; 388: 829–4042. . Weimar T Schena S, Bailey MS, et al. The Cox-maze procedure for lone atrial fibrillation: a single-center experience over 2 decades. CircArrhythmElectrophysiol. 2012;5:8–14.

43. Henn MC, Lancaster TS, Miller JR, et al. Late outcomes after the Cox maze IV procedure fo ratrial fibrillation. J Thorac Cardiovasc Surg. 2015;150:1168–1176

44. Benjamin EJ. Status of the epidemiology of atrial fibrillation.MedClin North Am. 2008 Jan;92(1):17-40, ix.

45. The Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) Investigators. N Engl J Med 2002; 347:1825-1833

46. Kannel WB (Feb 1976). "Some lessons in cardiovascular epidemiology from Framingham". Am J Cardiol. 37 (2): 269–82.

47. Morin DP[1], Bernard ML[2], Madias C[3], Rogers PA[2], Thihalolipavan S[2], Estes NA 3rd[4]. The State of the Art: Atrial Fibrillation Epidemiology, Prevention, and Treatment.Mayo Clin Proc. 2016 Dec;91(12):1778-1810. doi: 10.1016/j.mayocp.2016.08.022. Epub 2016 Nov 5.

48. Samuel Lévy et al Characterization of Different Subsets of Atrial Fibrillation in General Practice in France, 15 Jun 1999Circulation. 1999;99:3028–3035

49. Auer J, Scheibner P, Mische T, et al. Subclinical hyperthyroidism as a risk factor for atrial fibrillation. Am Heart J. 2001;142:838–842. [PubMed]

50. Barbisan JN, Fuchs FD, D'AgordSchaan B. Prevalence of thyroid dysfunction in patients with acute atrial fibrillation attended at a cardiology emergency room. Sao Paulo Med J 2003; 121 (4): 159-62.

51. Tischler MD, Le TH, Andrew KAM, et al. Clinical echocardiographic and doppler correlates of clinical instability with onset of atrial fibrillation. Am J Cardiol1990;66:721-24.

52. Flaker GC, Fletcher KA, Rothbart RM, et al. Clinical and echocardiographic features of intermittent atrial Fibrillation that predict recurrent atrial fibrillation. Am J Cardiol1995;76:355-358.

53. Wood P.1954, An appreciation of mitral stenosis. Brit Med J 1954; 1051. 12.

54. Deverall PB, Olley PM, Smith DR, Watson DA, and Whitaker W. Incidence of systemice. (1964). Embolism before and after mitral valvotomy. Thorax 1968; 23:530.

55. Teicholz E, Haft I, Atrial Fibrillation, Left ventricular Hypertrophy, Left atrial Enlargement, Ejection fraction, And Hypertension Inpatients With Nonhemorrhagic stroke Chest. 2007;132(4_MeetingAbstracts):578a. doi:10.1378/chest.132.4_MeetingAbstracts.578a

56. JieXue,[1] Yuanshao Lin,[1] Wensi Huang,[1,2] Xiaoli Chen,[1] Qian Li,[1] Zhengyi Cai,[1] Wanli Zhang,[1] Yuansheng Ye,[3] and Bei Shao[1] Left atrial size and risk of recurrent ischemic stroke in a Chinese populationBrain Behav. 2017 May; 7(5): e00702.

57. Mahmood ul Hassan[1], Hussain C, Gul AM, ullah Jan H, Hafizullah M. Frequency of left atrial and appendage clot in patients with severe mitral stenosis.J Ayub Med Coll Abbottabad. 2010 Apr-Jun;22(2):40-2.

ANNEXURES

PROFORMA

Clinical and Echocardiographic profile of Atrial Fibrillation

Name :

Age/Sex :

OP/IP No :

Occupation :

Address :

Contact No. :

SYMPTOMS

- ◊ Palpitations
- ◊ Dyspnea
- ◊ Chest pain
- ◊ Syncope
- ◊ Cough
- ◊ Hemoptysis
- ◊ Leg swelling
- ◊ Weakness of limbs
- ◊ Bleeding manifestaions
- ◊ Dysphagia

Patient Characteristics

- ◊ Smoker
- ◊ Alcoholic
- ◊ Diabetic
- ◊ Hypertensive
- ◊ Coronary Artery Disease

- ◊ Stroke
- ◊ Bronchial Asthma/COPD
- ◊ Thyroid disorder
- ◊ H/o cardiac surgery

GENERAL EXAMINATION :

VITALS :

SYSTEMIC EXAMINATION

CVS:

RS:

P/A

CNS:

INVESTIGATIONS:

TFT

PT/INR

ECG

Chest XRAY

ECHO

Others :

DIAGNOSIS:

INFORMATION SHEET

We are conducting a study on **"Clinical and Echocardiographic profile of Atrial Fibrillation"** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is to analyse the clinical, echocardiographic profile of Atrial fibrillation cases in medical ward and OPD. We are selecting certain cases and if you are found eligible, we may perform extra tests and special studies which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator | Signature of Participant

Date :

Place :

PATIENT CONSENT FORM

Study Detail	:	**Clinical and Echocardiographic profile of Atrial Fibrillation**
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests. ☐

Signature/thumb impression
Patient's Name and Address

Signature of Investigator
Dr.G. AISHWARYA.

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To

Dr.G.Aishwarya
I Year PG in MD General Medicine
Institute of Internal Medicine
Madras Medical College
Chennai 600 003

Dear Dr.G.Aishwarya,

The Institutional Ethics Committee has considered your request and approved your study titled **"CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF ATRIAL FIBRILLATION " - NO.15062017(A)**

The following members of Ethics Committee were present in the meeting hold on **20.06.2017** conducted at Madras Medical College, Chennai 3

1. Prof.Dr.C.Rajendran, MD.,	:Chairperson
2. Prof.R.Narayana Babu,MD.,DCH., MMC,Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	:Member Secretary
4. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3	: Member
5. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC	: Member
6. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai	: Member
7. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3	: Member
8.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
9.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist
10.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person

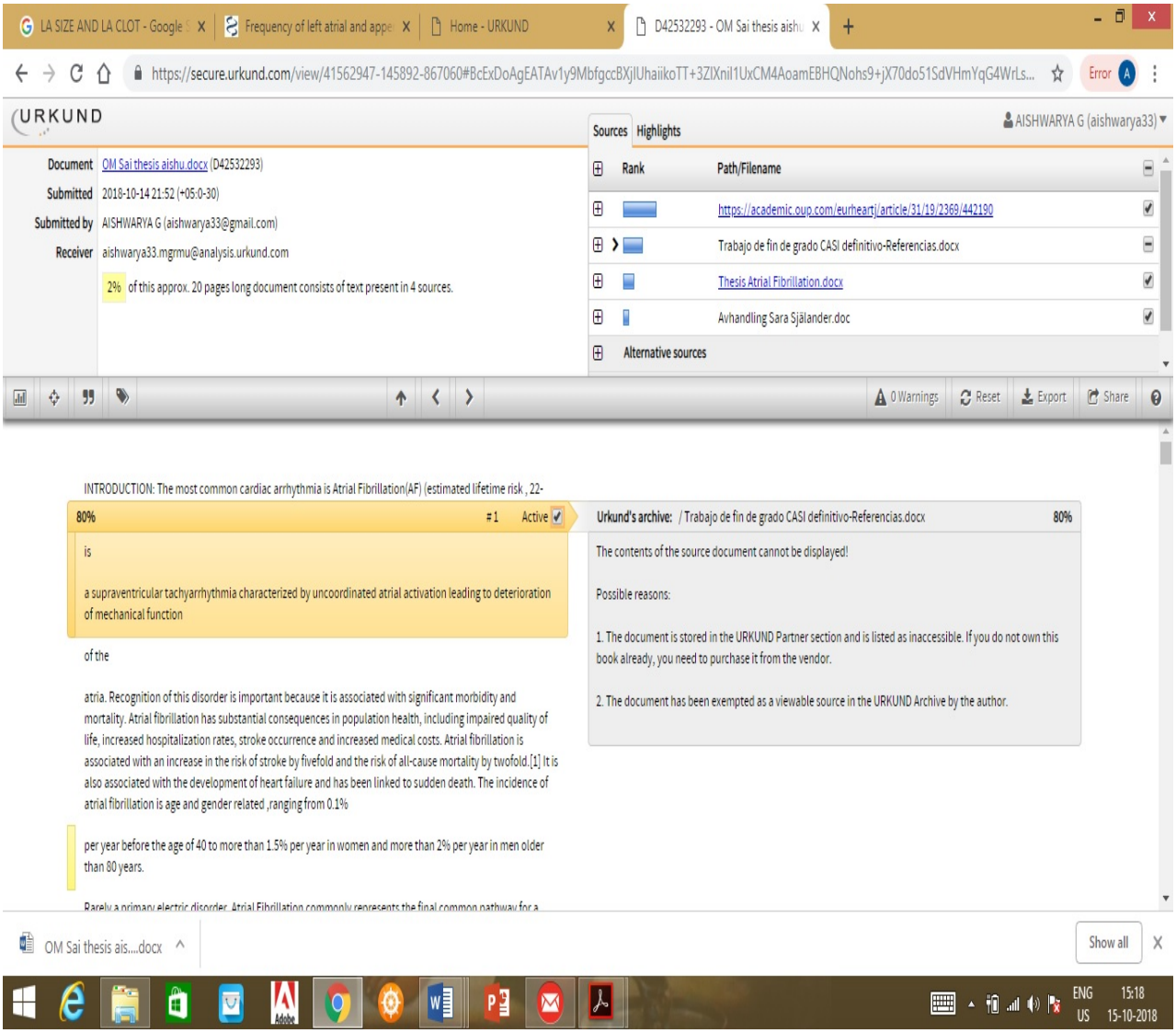
We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary - Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

PLAGIARISM DIGITAL RECEIPT



PLAGIARISM SCREENSHOT

URKUND

Urkund Analysis Result

Analysed Document:	OM Sai thesis aishu.docx (D42532293)
Submitted:	10/14/2018 6:22:00 PM
Submitted By:	aishwarya33@gmail.com
Significance:	2 %

Sources included in the report:

Thesis Atrial Fibrillation.docx (D42392000)
Avhandling Sara Själander.doc (D21404665)
Trabajo de fin de grado CASI definitivo-Referencias.docx (D26430518)
https://academic.oup.com/eurheartj/article/31/19/2369/442190

Instances where selected sources appear:

9

CERTIFICATE – II

This is to certify that this dissertation work titled **"CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF ATRIAL FIBRILLATION"** of the candidate **Dr. G.AISHWARYA** with registration Number **201611001** for the award of **M.D.** in the branch of **GENERAL MEDICINE**. I personally verified the urkund.com website for plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **2 percentage** of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

AGE	SEX	FATIGUE	DYSPNEA	PALPIT	SYNCOPE	CHEST P	ASYMP	mod EHRA score	BMI CATEGORY	ETIOLOGY	LESION	CVA(EMBOLIC)	PVD	DM	HT RATE	INR	CHA2DS2VASc	EF	LA SIZE (CM	LA SIZECAT	AUTOCONTRA	CLOT	LVSD	LVDD	LVH	BLEEDING MANIF
18	F	Y	III	Y	N	Y	N	3	UNDERWEIGHT	RHD	MS,MR	N	N	N	145	2.00	NA	55%	5.00	>4 - 5	Y	N	NORMAL	NIL	N	N
18	F	Y	II	Y	N	N	N	2b	NORMAL	RHD	MS,MR	N	N	N	100	2.50	NA	55%	4.50	>4 - 5	N	N	NORMAL	NIL	N	N
19	M	Y	III	N	N	Y	N	3	NORMAL	RHD	MS	N	N	N	145	1.80	NA	65%	5.00	>4 - 5	N	LA CLOT	NORMAL	NIL	N	N
19	F	N	II	N	N	N	N	2b	UNDERWEIGHT	RHD	MS	N	N	N	100	2.50	NA	65%	4.50	>4 - 5	N	N	NORMAL	NIL	N	N
21	M	N	I	N	Y	N	N	2a	NORMAL	RHD	MS,AS,AR	N	N	N	100	2.50	NA	65%	5.50	> 5 - 6	N	N	NORMAL	NIL	Y	N
23	M	N	II	Y	Y	Y	N	2b	NORMAL	RHD	MS,AS,AR	N	N	N	120	2.50	NA	50%	4.20	>4 - 5	N	N	NORMAL	NIL	Y	N
24	F	N	II	N	N	N	N	2b	NORMAL	RHD	MS	N	N	N	100	2.00	NA	65%	4.10	>4 - 5	N	N	NORMAL	NIL	N	N
25	M	N	I	N	N	N	N	2a	UNDERWEIGHT	RHD	MS	N	N	N	110	2.00	NA	65%	4.20	>4 - 5	N	N	NORMAL	NIL	N	N
27	F	Y	IV	Y	N	Y	N	4	UNDERWEIGHT	RHD	MS,MR	Y	N	N	140	1.50	NA	55%	5.20	> 5 - 6	N	LA CLOT	NORMAL	NIL	N	N
27	F	Y	IV	N	N	Y	N	4	NORMAL	RHD	MS	N	Y	N	140	1.70	NA	65%	5.20	> 5 - 6	N	LA CLOT	NORMAL	NIL	N	N
27	F	Y	IV	Y	N	N	N	4	UNDERWEIGHT	RHD	MS	N	N	N	90	2.00	NA	65%	5.00	>4 - 5	N	N	NORMAL	NIL	N	N
28	M	N	III	Y	N	N	N	3	NORMAL	RHD	MS,MR,AS,AR	N	N	N	90	2.00	NA	60%	4.90	>4 - 5	N	N	NORMAL	NIL	Y	N
28	M	N	N	N	Y	N	N	4	NORMAL	LONE AF	N	N	N	N	120	1.50	2	65%	3.90	< 4 CM	N	N	NORMAL	NIL	N	N
30	M	N	II	Y	N	N	N	2b	NORMAL	HTN	CONC LVH	N	N	N	95	2.00	2	60%	4.50	>4 - 5	N	N	NORMAL	GR II	Y	N
30	M	N	II	N	N	N	N	2b	NORMAL	HTN	CONC LVH	N	N	N	90	2.00	2	60%	4.20	>4 - 5	N	N	NORMAL	GR II	Y	N
30	F	Y	III	N	N	Y	N	3	NORMAL	DCM	GLOBAL HYPOKIN	N	N	N	95	2.30	3	25%	4.40	>4 - 5	N	N	SEVERE	NIL	N	N
31	F	N	II	N	N	N	N	2b	MORBIDLY OBESE	HTN	CONC LVH	N	N	N	100	2.00	2	60%	4.10	>4 - 5	N	N	NORMAL	GR II	Y	N
31	M	Y	III	N	N	Y	N	3	NORMAL	CAD,HTN	MILD LVSD	N	N	N	95	1.80	4	38%	4.40	>4 - 5	N	N	MILD	GR I	N	N
32	F	N	II	N	Y	N	N	4	MORBIDLY OBESE	CAD	SEVERE LVSD	Y	N	N	98	2.00	5	28%	5.50	> 5 - 6	N	LV CLOT	SEVERE	NIL	N	N
32	F	N	II	Y	N	N	N	3	NORMAL	RHD	MS,MR	N	Y	N	115	1.50	NA	55%	5.00	>4 - 5	N	LA CLOT	NORMAL	NIL	N	N
32	F	N	II	Y	N	N	N	2b	NORMAL	RHD	MS	N	Y	N	120	1.30	NA	60%	5.50	> 5 - 6	N	LA CLOT	NORMAL	NIL	N	N
33	M	N	I	N	N	N	N	2a	NORMAL	RHD	MS	N	N	N	90	2.00	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	N
33	M	N	I	N	N	N	N	2a	NORMAL	RHD	MS,AS,AR	N	N	N	95	2.20	NA	58%	4.80	>4 - 5	N	N	NORMAL	NIL	Y	N
35	M	Y	II	Y	Y	Y	N	2b	UNDERWEIGHT	RHD	MS,AS,AR	N	N	N	114	1.90	NA	55%	5.00	>4 - 5	N	N	NORMAL	GR I	Y	N
35	M	N	II	N	N	N	N	2a	NORMAL	RHD	MS	N	N	N	100	2.00	NA	55%	5.00	>4 - 5	N	N	NORMAL	NIL	N	N
35	F	N	N	Y	N	N	N	2a	UNDERWEIGHT	OVERT HYPERT	N	N	N	N	120	1.00	0	65%	5.50	> 5 - 6	N	N	NORMAL	NIL	N	N
36	M	N	N	Y	N	N	N	2a	UNDERWEIGHT	OVERT HYPERT	N	N	N	N	118	1.10	0	60%	3.90	< 4 CM	N	N	NORMAL	NIL	N	N
36	M	N	N	Y	N	N	N	2a	UNDERWEIGHT	OVERT HYPERT	N	N	N	N	113	1.10	0	60%	3.90	< 4 CM	N	N	NORMAL	NIL	N	N
38	F	N	N	Y	N	N	N	2a	UNDERWEIGHT	OVERT HYPERT	N	N	N	N	115	1.20	0	65%	3.80	< 4 CM	N	N	NORMAL	NIL	N	N
38	F	N	N	Y	N	N	N	2a	UNDERWEIGHT	OVERT HYPERT	N	N	N	N	112	1.30	0	60%	3.80	< 4 CM	N	N	NORMAL	NIL	N	N
39	M	N	N	Y	N	N	N	2a	UNDERWEIGHT	OVERT HYPERT	N	N	N	N	110	1.30	0	65%	3.90	< 4 CM	N	N	NORMAL	NIL	N	N
40	F	N	N	Y	N	N	N	2a	UNDERWEIGHT	OVERT HYPERT	N	N	N	N	130	1.00	0	65%	3.80	< 4 CM	N	N	NORMAL	NIL	N	N
40	F	N	II	N	N	N	N	2b	MORBIDLY OBESE	OVERT HYPOT	PERICARD EFFUSION	N	N	N	100	1.10	0	50%	3.50	< 4 CM	N	N	NORMAL	NIL	N	N
40	M	N	II	N	N	N	N	2b	MORBIDLY OBESE	OVERT HYPOT	PERICARD EFFUSION	N	N	N	98	1.30	0	50%	3.50	< 4 CM	N	N	NORMAL	NIL	N	N
40	F	N	N	N	N	N	Y	1	UNDERWEIGHT	SUBCL HYPERT	N	N	N	N	95	1.20	0	65%	3.70	< 4 CM	N	N	NORMAL	NIL	N	N
40	F	N	N	N	N	N	Y	1	UNDERWEIGHT	SUBCL HYPERT	N	N	N	N	90	1.00	0	65%	3.80	< 4 CM	N	N	NORMAL	NIL	N	N
41	M	N	I	N	N	N	N	2a	NORMAL	HTN	CONC LVH	N	N	N	95	1.60	2	60%	3.90	< 4 CM	Y	N	NORMAL	GR I	Y	N
43	M	N	I	N	N	N	N	2a	NORMAL	HTN	CONC LVH	N	N	N	98	1.70	1	60%	3.90	< 4 CM	N	N	NORMAL	GR I	Y	N
43	F	Y	III	Y	N	Y	N	3	MORBIDLY OBESE	HTN	CONC LVH	Y	N	N	110	1.40	6	55%	5.50	> 5 - 6	Y	N	NORMAL	GR III	Y	N
43	M	N	N	N	N	N	Y	1	OBESE 1	HTN	CONC LVH	N	N	N	98	1.90	3	55%	4.00	>4 - 5	N	N	NORMAL	GR I	Y	N
43	M	N	N	N	N	N	Y	1	PRE OBESE	HTN	CONC LVH	N	N	N	90	2.00	3	55%	4.50	>4 - 5	N	N	NORMAL	GR I	Y	N
44	M	N	II	N	N	N	N	2b	UNDERWEIGHT	RHD	MS	N	N	N	130	2.00	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	N
45	F	N	II	N	N	N	N	2b	UNDERWEIGHT	RHD	MS	N	N	N	113	2.00	NA	60%	4.90	>4 - 5	N	N	NORMAL	NIL	N	N
45	M	N	II	N	N	N	N	2b	NORMAL	RHD	MS	N	N	N	114	2.00	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	N
45	M	N	II	N	Y	N	N	2b	NORMAL	RHD	MS,AS,AR	N	N	N	105	2.10	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	Y	N
45	F	N	II	N	N	N	N	2b	NORMAL	HTN	CONC LVH	N	N	N	100	2.30	3	55%	5.00	>4 - 5	N	N	NORMAL	GR I	Y	N
48	M	N	I	N	N	N	N	2a	NORMAL	HTN	CONC LVH	N	N	N	98	1.80	1	60%	4.50	>4 - 5	N	N	NORMAL	GR I	Y	N
48	M	N	N	N	N	N	Y	1	PRE OBESE	HTN	CONC LVH	N	N	Y	98	2.10	3	60%	4.90	>4 - 5	N	N	NORMAL	GR I	Y	N
48	F	N	II	N	N	N	N	2a	MORBIDLY OBESE	HTN	CONC LVH	N	N	Y	99	1.30	4	55%	5.00	>4 - 5	Y	N	NORMAL	GR II	Y	N
50	F	N	II	N	N	Y	N	2b	NORMAL	HTN	CONC LVH	N	N	Y	112	1.30	5	55%	6.00	> 5 - 6	N	LA CLOT	NORMAL	GR II	Y	N
50	F	N	II	N	N	Y	N	2b	NORMAL	HTN	CONC LVH	N	N	Y	111	1.20	5	58%	6.50	>6	Y	N	NORMAL	GR II	Y	N
50	M	Y	III	Y	N	Y	N	3	MORBIDLY OBESE	HTN	CONC LVH	N	N	Y	141	2.10	2	55%	6.00	> 5 - 6	N	N	NORMAL	GR III	Y	N
53	M	N	I	N	N	N	N	2a	MORBIDLY OBESE	HTN	CONC LVH	Y	N	Y	98	1.50	5	55%	5.50	> 5 - 6	N	N	NORMAL	GR II	Y	N
53	F	N	II	N	N	N	N	2a	OVERWEIGHT	HTN	CONC LVH	Y	N	Y	105	1.60	4	55%	5.00	>4 - 5	Y	N	NORMAL	GR II	Y	N
53	M	N	I	N	N	N	N	2a	MORBIDLY OBESE	HTN	CONC LVH	N	N	Y	96	1.70	2	60%	4.80	>4 - 5	Y	N	NORMAL	GR I	Y	N
54	M	Y	III	Y	N	Y	N	3	NORMAL	HTN	CONC LVH	N	N	Y	135	2.10	3	55%	5.00	>4 - 5	N	N	NORMAL	GR III	Y	N
55	M	Y	III	N	N	Y	N	3	OBESE	CAD	MOD LVSD	N	N	N	120	2.10	1	36%	5.00	>4 - 5	N	N	MOD	NIL	N	N
55	M	Y	III	Y	N	Y	N	3	NORMAL	CAD	SEVERE LVSD	N	N	Y	112	2.00	2	28%	5.40	> 5 - 6	N	N	SEVERE	NIL	N	N
55	M	N	I	N	N	N	N	2a	NORMAL	CAD	MILD LVSD	N	N	Y	96	2.10	3	41%	4.90	>4 - 5	N	N	MILD	NIL	N	N
55	M	Y	II	Y	N	N	N	2a	OBESE	CAD	SEVERE LVSD	Y	N	Y	141	1.70	5	25%	5.00	>4 - 5	Y	N	SEVERE	NIL	N	N
55	F	N	II	N	N	N	N	2a	NORMAL	CAD	MILD LVSD	N	N	N	96	2.00	3	42%	4.70	>4 - 5	N	N	MILD	NIL	N	N
55	F	N	III	Y	N	Y	N	3	OBESE	CAD	MOD LVSD	N	N	Y	120	2.10	3	34%	4.80	>4 - 5	N	N	MOD	NIL	N	N
56	M	Y	IV	Y	N	Y	N	4	MORBIDLY OBESE	CAD	SEVERE LVSD	N	N	N	134	2.10	1	24%	5.50	> 5 - 6	Y	N	SEVERE	NIL	N	N
57	F	Y	IV	Y	N	Y	N	4	OBESE	CAD	SEVERE LVSD	Y	N	Y	130	1.60	4	28%	5.00	>4 - 5	N	LA CLOT	SEVERE	NIL	N	N
58	F	N	II	N	N	N	N	2b	MORBIDLY OBESE	CAD	MILD LVSD	N	N	Y	121	2.00	2	41%	4.80	>4 - 5	Y	N	MILD	NIL	N	N
59	M	Y	III	Y	N	N	N	3	OBESE	CAD	SEVERE LVSD	Y	N	Y	145	1.70	5	26%	5.00	>4 - 5	N	LA CLOT	SEVERE	NIL	N	N

60	F	N	III	Y	N	N	N	2b	OVERWEIGHT	CAD	SEVERE LVSD	Y	N	N	148	1.50	4	24%	5.50	> 5 - 6	N	LV CLOT	SEVERE	NIL	N	N
60	F	N	III	Y	N	N	N	3	NORMAL	RHD	MS	N	N	N	120	4.50	NA	60%	4.50	>4 - 5	N	N	NORMAL	NIL	N	ICH
60	F	N	I	N	N	N	N	2a	UNDERWEIGHT	RHD	MS,MR	N	N	N	98	4.00	NA	60%	5.50	> 5 - 6	N	N	NORMAL	NIL	N	ECCHYMOSES
61	F	N	II	N	N	N	N	2b	NORMAL	RHD	MS	N	Y	N	97	1.70	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	N
61	M	N	N	N	N	N	N	1	NORMAL	RHD	MS	Y	N	N	99	1.80	NA	60%	5.50	> 5 - 6	N	LA CLOT	NORMAL	NIL	N	N
61	F	N	II	Y	N	N	N	2a	NORMAL	RHD	MS,MR	N	N	N	140	5.10	NA	55%	6.00	> 5 - 6	N	N	NORMAL	NIL	N	ICH
62	F	N	II	N	N	N	N	2a	NORMAL	RHD	MS	N	Y	N	131	2.00	NA	60%	5.50	> 5 - 6	N	LA CLOT	NORMAL	NIL	N	N
65	F	N	N	N	N	N	N	1	UNDERWEIGHT	RHD	MS	N	Y	N	133	1.70	NA	60%	5.50	> 5 - 6	N	LA CLOT	NORMAL	NIL	N	N
65	F	N	N	N	N	N	N	1	NORMAL	RHD	MS	Y	N	N	96	2.00	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	N
65	F	N	N	N	N	N	N	1	MORBIDLY OBESE	S/P MVR	S/P MVR	N	N	N	94	3.50	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	GUM BLEED
66	F	N	II	Y	N	N	N	2a	NORMAL	RHD	MS,MR	N	N	N	114	1.50	NA	60%	5.50	> 5 - 6	Y	N	NORMAL	NIL	N	N
66	M	N	IV	Y	N	N	N	4	NORMAL	RHD	MS,MR	N	N	N	121	2.00	NA	50%	6.00	> 5 - 6	N	N	NORMAL	NIL	N	N
66	M	N	I	N	Y	Y	N	2a	NORMAL	HCM	HCM	N	N	N	90	1.20	0	60%	4.50	>4 - 5	N	N	NORMAL	GR II	Y	N
67	F	N	III	N	N	N	N	3	OVERWEIGHT	S/P MVR	SEVERE LVSD	N	N	Y	94	4.10	NA	28%	5.50	> 5 - 6	N	N	SEVERE	NIL	N	EPISTAXIS
67	F	N	III	Y	N	N	N	3	NORMAL	S/P MVR	N	N	N	N	112	4.50	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	HEMATEMESIS
68	M	N	III	Y	N	N	N	3	NORMAL	COPD COR P	RA,RV DIL, RVD	N	N	N	130	2.00	1	60%	5.50	> 5 - 6	N	N	NORMAL	NIL	N	N
68	M	N	I	Y	N	N	N	2a	NORMAL	RHD	MS,MR	N	N	N	120	2.00	NA	65%	5.50	> 5 - 6	N	N	NORMAL	NIL	N	N
68	F	N	I	N	N	N	N	2a	NORMAL	RHD	MS	N	N	N	145	5.00	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	ICH
68	F	N	I	N	N	N	N	2a	OVERWEIGHT	CTEPH	RA,RV DIL, RVD	N	N	Y	150	5.00	3	60%	4.50	>4 - 5	N	N	NORMAL	NIL	N	CEREBELLAR BLEED
69	M	N	II	Y	N	N	N	2b	NORMAL	RHD	MS,MR	Y	N	N	136	2.10	NA	65%	5.50	> 5 - 6	Y	N	NORMAL	NIL	N	N
70	M	N	IV	Y	N	Y	N	4	NORMAL	CAD	SEVERE LVSD	N	N	N	136	1.60	2	28%	5.00	>4 - 5	N	N	SEVERE	NIL	N	N
71	F	N	III	Y	N	N	N	3	UNDERWEIGHT	RHD	S/P MVR	N	N	N	150	1.90	NA	60%	5.50	> 5 - 6	N	PR V CLOT	NORMAL	NIL	N	N
71	M	N	N	N	N	N	N	1	NORMAL	DCM	MOD LVSD	Y	N	N	89	1.30	1	39%	4.00	>4 - 5	N	LV CLOT	MOD	NIL	N	N
73	F	N	II	Y	N	N	N	2a	NORMAL	RHD	MS	N	N	N	110	4.50	NA	60%	5.00	>4 - 5	N	N	NORMAL	NIL	N	SDH
73	F	N	IV	Y	N	N	N	4	OVERWEIGHT	CAD	MOD LVSD	N	N	Y	141	1.80	3	38%	5.00	>4 - 5	N	N	MOD	NIL	N	N
74	M	N	N	Y	N	N	N	2a	NORMAL	RHD	MS,MR	N	N	N	86	2.10	NA	65%	5.50	> 5 - 6	N	N	NORMAL	NIL	N	N
74	M	N	IV	Y	N	Y	N	4	NORMAL	CAD	SEVERE LVSD	Y	N	Y	150	2.00	3	26%	5.00	>4 - 5	N	N	SEVERE	NIL	N	N
77	F	N	III	N	N	N	N	3	OVERWEIGHT	OVERT HYPOT	PERICARD EFFUSION	N	N	N	120	2.00	0	60%	4.00	>4 - 5	N	N	NORMAL	NIL	N	N
78	F	N	II	N	N	N	N	2b	UNDERWEIGHT	RHD	MS	N	N	N	100	2.50	NA	65%	4.10	>4 - 5	N	N	NORMAL	NIL	N	N
80	M	Y	III	N	N	Y	N	3	NORMAL	CAD,HTN	MILD LVSD	N	N	N	95	1.80	4	38%	4.40	>4 - 5	N	N	MILD	GR I	N	N
80	M	N	I	N	Y	N	N	2a	NORMAL	RHD	MS,AS,AR	N	N	N	95	2.20	NA	58%	4.80	>4 - 5	N	N	NORMAL	NIL	Y	N
80	F	Y	III	Y	N	Y	N	3	UNDERWEIGHT	RHD	MS,MR	N	N	N	145	2.00	NA	55%	5.00	>4 - 5	Y	N	NORMAL	NIL	N	N
81	M	N	IV	Y	N	N	N	4	NORMAL	RHD	MS,MR	N	N	N	121	2.00	NA	50%	6.00	> 5 - 6	N	N	NORMAL	NIL	N	N
69	M	N	II	Y	N	N	N	2b	NORMAL	RHD	MS.MR	Y	N	N	136	2.10	NA	65%	5.50	> 5 - 6	Y	N	NORMAL	NIL	N	N